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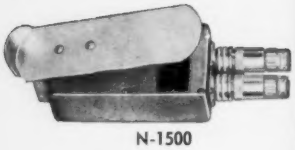

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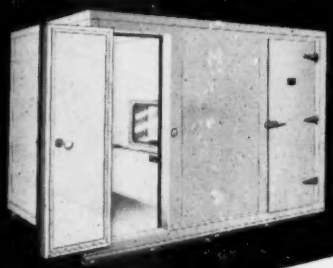

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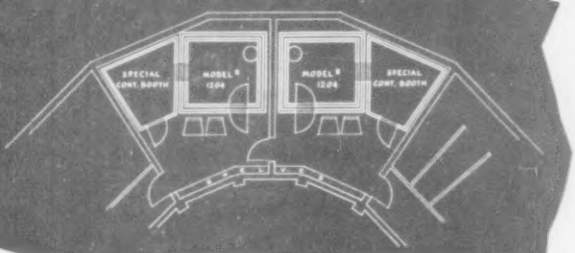

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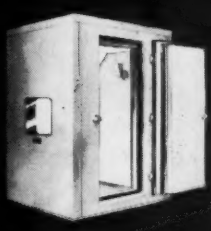

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"Necrophonia"

KURT TSCHIASSNY, M.D., Cincinnati

You will not find the word "necrophonia" in any medical dictionary. I coined it to designate the laryngeal sounds which may appear under certain conditions as a spontaneous postmortem phenomenon. This may also be experimentally produced by certain manipulations of the dead larynx. A suitable subtitle of this paper might be "A myth is blasted" or "A ghost is unmasked." You will shortly understand the meaning of these phrases.

The idea of performing the experiments, which will be demonstrated, occurred to me about 12 years ago while preparing a paper which dealt with the history of the tuning fork tests.[1] At this occasion I learned that Dr. Friedrich Hofmann, a general practitioner in Burgsteinfurt, a small place in Northern Germany, invented the perforated light reflector.[2] From his biography[3] I learned that he also was the author of an article which appeared in 1847 under the title "A Simple Method of Preventing Burial While Still Alive." Unfortunately this article is not available to me. The existence of such a paper indicates that being buried alive was regarded as a reality occurring frequently enough to justify elaboration of methods for its prevention. Moreover, it seems that mystery stories of E. Th. A Hoffmann, Edgar Allen Poe, and other "Alfred Hitchcocks" of the 19th century added fuel to the fire of a nightmare which at that time was horrifying minds all over the world, eventually advancing to a pandemic anxiety neurosis. I remember even when I was an intern in Vienna in the beginning of the century that we sometimes had to perform cardiac puncture in accordance with the last will of the deceased or as requested by the relatives. The cause for this neurosis was the occasional appearance of the voice of the dead person sounding through the closed coffin while resting in the chapel or after burial in the graveyard.

The title of Hofmann's article stimulated me to perform experiments by which the possibility of phonation in the dead larynx was shown. I presented them for the first time at one of our staff conferences several years ago and later repeatedly in clinical courses. I considered that such experiments may provide a possible explanation for this terrifying postmortem phenomenon.

The Larynx as a Musical Instrument

It appears necessary to preface the presentation of these experiments with a few remarks concerning the principles of laryngeal voice. The larynx viewed as a sound-producing instrument belongs to the category of the "cushion whistle," or "Polsterpfeife," as it was called by Helmholtz.[4] The sound-producing mechanism of a cushion whistle depends principally on two factors:

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From the Department of Otolaryngology College of Medicine, University of Cincinnati.

Presented at the 33d Annual Convention of the American Speech and Hearing Association, Nov. 22, 1957.

1. An elastic structure encircling in a cushion-like fashion a central gap. This opening can be closed tightly by a muscular sphincter.

2. Gas pressure directed toward the closed gap. When the pressure overcomes the sphincter force, the gap is opened, allowing the escape of gas. With the consequent and immediate drop of pressure the sphincter again becomes effective and closes the gap. This cycle is repeated. The repetitive alternate closure and opening of the gap is the factor responsible for interruption of the gas column. The vibrating body in this type of instrument is represented predominantly by the gas column and only to a minor degree (if at all) by the cushion. It is not the alternate contraction and relaxation of any muscle.

In the laryngeal cushion whistle the active gas is air; the cushion is represented by the vocal lips; the gas is called "glottis," and the sphincter action is provided by the system of the adductor and tensor muscles.

Some other examples of cushion whistle are the following: 1. The lips of the horn-blower and trumpeter. The horn per se or the tube of the trumpet or any analogous brass instruments is responsible only for amplification and modification of the air column which is forced through the gap formed by the lips. The lips are tightly closed by the orbicularis oris which is supplied by the facial nerve. One may understand now why in Bell's palsy the patient cannot whistle. 2. The cricopharyngeal sphincter opened by the eruption of gases accumulated in the stomach, particularly air or CO_2, resulting in audible belching. 3. The pseudoglottis in the laryngectomized patient. 4. The anal cushion whistle activated when the sphincter ani is forced open by gases produced by gas-forming bacilli, particularly the Escherichia coli (Bacterium coli).

In man and the majority of mammals expiration is the active phase in phonation. However, some particularly talkative persons may use inspiration as well in order not to lose time. Ventriloquists, when presenting the fictional voice of the dummy, allegedly use inspiratory phonation in addition to some tricks. In the cat inspiration is the phase activating phonation, according to DuBois-Raymond and Katzenstein. These authors provoked phonation by stimulation of the ala cinerea, the medullary center for inspiration. This is contrary to experiments with other animals, where stimulation of the calamus scriptorius, the medullary center for expiration, caused this effect. The donkey, according to Grützner, for his "E—Ah" uses both phases; inspiration for "E" and expiration for the "Ah."[5]

Models

For a better understanding of the rather complicated and still controversial functional anatomy of the laryngeal musculature two working models may be helpful. We constructed them many years ago and used them for teaching in our clinical courses.

Model 1.—The first model represents a transverse section through the larynx at the level of the vocal cords. You see a curved bar angulated anteriorly which represents the thyroid cartilage, and a transverse bar representing the cricoid lamina. Two triangular pieces upon the lamina and freely articulating with it represent the two arytenoids. Extending from the anterior tip of the arytenoids to the thyroid angle, are the vocal cords, indicated by two pieces of rubber tubing. Furthermore, you see several ribbons, red ones and green ones. The former represent the adductor muscles, the latter the two abductors. The adductors consist of an anterior portion formed by the cricothyroarytenoid muscular system and a posterior portion between the two arytenoids, called the interarytenoideus.

Action of the Laryngeal Muscles: The laryngeal muscles are generally classified as including three groups: the abductors, the adductors, and the tensors. The first model demonstrates the action of the abductors and adductors.

Fig. 1.—Glottis in quiet respiration. Both adductors and abductors are relaxed. Vocal cords in intermediary position.

Fig. 2.—Glottis in deep inspiration. Adductors are relaxed; abductors are contracted. Vocal cords in the lateral position.

1. During quiet respiration all laryngeal muscles are relaxed (Fig. 1). The glottis is slightly opened. The vocal cords are in a position about half-way between the midline and the lateral position. It is called the intermediary position of quiet respiration.

2. In deep inspiration, however (Fig. 2), when there is increased demand for air, as in exertion, high altitude, in anemias, in heart and lung diseases, etc., the glottis is widened. It occurs as a result of the contraction of the abductor muscles, the posterior cricoarytenoidei or more simply, the two posticus muscles. This changes the position of the vocal cords from the intermediary (respiratory) to the lateral (inspiratory) position. It must be emphasized that the posticus is not the opener but the dilator of the glottis. There does not exist a muscle with the function of opening the glottis. It opens spontaneously by relaxation of the entire laryngeal musculature. Paralysis of the abductors experimentally produced by Grossmann, Kuttner, and Katzenstein, Stupka, Herzog, and others, brings the vocal cords to the intermediary

Fig. 3.—Glottis in functional aphonia due to "transversus paralysis." Abductors and posterior adductors are relaxed; anterior adductors are contracted.

Fig. 4.—Glottis in functional aphonia due to "internus paralysis." Abductors and anterior adductors are relaxed; posterior adductors are contracted.

position. Calling midline fixation of the vocal cords "abductor paralysis" is a misnomer resulting from the famous but still controversial concept of Sir Felix Semon. Unfortunately this misleading term is still in common use. It seems that inherited malformations, even those of intellectual character, can hardly be eradicated. The matter is extensively discussed in a recent article, where pertinent references can be found.[6]

3. The adductors or sphincter muscles approximate the vocal cords. As mentioned already these muscles consist of an anterior and a posterior portion. Contraction of the anterior portion closes the membranous part of the glottis (Fig. 3), while the cartilaginous part presents an open triangular gap. Contraction of the posterior portion closes the cartilaginous part of the glottis while the membranous glottis is kept open, presenting an ovoid gap (Fig. 4). In our professional slang we call the former condition "transversus paralysis"; the latter is known as "internus paralysis." This term is probably not quite adequate since the ovoid shape of the glottis may be due to a functional relaxation of the posterior cricoarytenoid ligament. Either of these two positions may be seen in the whispering voice.

4. In phonation the entire glottis must be shut completely or almost completely. This is brought about by the combined action of the anterior and posterior groups of the laryngeal adductors. Thus the vocal cords assume the median or paramedian position which is called the midline or phonatory position (Fig. 5).

Model 2.—The action of the tensor muscles is demonstrated by the second model (Fig. 6). It represents a sagittal section through the larynx. You see the thyroid and the cricoid cartilages and the arytenoid cartilage with one vocal cord extending from the vocal process posteriorly to the thyroid angle anteriorly. The thyrocricoid articulation operated by the thyrocricoid muscle is distinctly shown, while the cricoarytenoid articulation is not demonstrated. In this model the action of this joint is assumed to be prevented as it occurs in reality by means of the posterior cricoarytenoid ligament. This structure is indicated by a piece of adhesive. Tension depends on the action of either one of two muscles, the cricothyroid and the inner thyroarytenoid muscle which is usually called the "internus" or "vocalis" muscle. Interestingly, these two muscles are mechanical antagonists but functionally they are synergists. Either one produces by its contrac-

Fig. 5.—Glottis in phonation. Abductors are relaxed; all the adductors are contracted. The entire glottis is closed, vocal cords in the (para-) median position.

Fig. 6.—Both tensor muscles are relaxed.

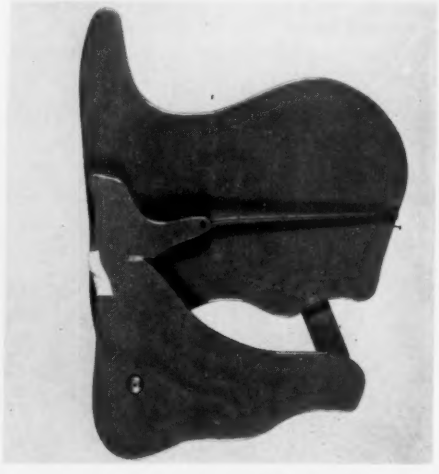

Fig. 7.—Tension produced by contraction of the cricothyroid muscle. Note elongation of the vocal cord.

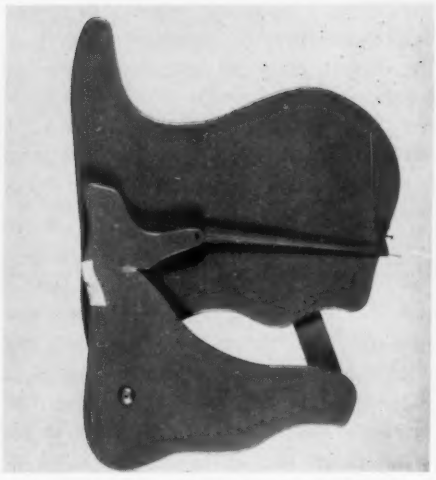

Fig. 8.—Tension produced by contraction of the vocalis muscle. Note shortening of the vocal cord.

tion increased tension of the vocal cords but in opposing ways. Contraction of the cricothyroid elongates the distance between the two points of attachment of the vocal cord (Fig. 7). This elongation must be compensated for by tension of the elastic vocal cord. Contrariwise, the vocalis muscle shortens the vocal cord without changing the distance of the points of attachment when the cricoarytenoid articulation is fixed by the posterior cricoarytenoid ligament as shown in the model (Fig. 8). Shortening of the vocal cords is compensated for by increased tension of the elastic vocal ligament.

Experimental Phonation in the Dead Larynx

As we learned from this rather extensive discussion, laryngeal phonation in the living mammal depends principally on two factors, namely, tight closure of the glottis by means of the adductor and tensor muscles, and blasting of the closed glottis by expiratory or inspiratory air pressure. Since these two factors are absent in the dead larynx, they are replaced in the experiment by digital pressure upon the lateral laryngeal walls and by gas pressure directed from a gas reservoir (oxygen tank) through the trachea toward the forcibly closed glottis.

This experiment will now be shown with the larynges of dogs, cats, pigs, and man. You can hear sounds which unmistakably resemble the natural voice of the individual larynx as it appears during life. In the human specimen, which is taken from a man of 23 who two days ago was the victim of a car accident, we can produce a series of sounds, including almost an octave, by appropriate increase or decrease of either the digital or the gas pressure. By means of these maneuvers laryngeal phonation as demonstrated in the dead mammal is not a surprising phenomenon. It is produced by a procedure which represents essentially an artificial duplication of the natural mechanism of phonation. I am sure that similar studies have been repeatedly performed by older investigators.

However, I still owe you an explanation for the voice emanating from the closed coffin where no manipulations are possible. The answer is simple and clear. Both requirements for laryngeal phonation may be met with under postmortem conditions. The phonatory position of the vocal cords appears regularly in rigor mortis, which per-

sists for a day or more. The gases responsible for the pressure which blasts the closed glottis are occasionally produced by gas-forming bacteria which may invade the air passages during the agonal or the postmortem period and accumulate underneath the tightly closed glottis. Thus necrophonia may be produced.

Conclusion

I anticipated your interest in these matters for three reasons:

1. The models are helpful in understanding and teaching functional anatomy of the laryngeal musculature and are readily duplicated.

2. The experiments may explain the mysterious laryngeal phonation noted in dead persons. The old horrifying nightmare of being buried alive is dissipated.

3. These experiments by application of tonometric, manometric, and stroboscopic devices may open the door to future studies concerning factors integrated in the mechanism of phonation.

101 Louis Ave. (20)

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Histoplasmosis Involving the Larynx

CAPT. HUESTON C. KING (MC), U. S. Army, and CAPT. JOHN F. X. CLINE (MC), U. S. A. F.

In the past decade and a half histoplasmosis has risen from relative obscurity to a prominent place in medical thinking. While almost all the tissues of the body may be affected by this disease, and it has come repeatedly under the eye of the otolaryngologist, only about 20 cases have been reported in which the larynx has been involved. A review of the literature leads one to suspect that laryngeal involvement may be a commoner problem than this small number of cases indicates. A brief review of the disease is accompanied by another case report of a patient with extensive laryngeal involvement.

Histoplasmosis is defined by Roberts [1] as a subacute or chronic infection caused by the fungus Histoplasma capsulatum.[1] Darling [2] first described the disease in 1905, when he discovered the causative organisms in the tissues. Because of their appearance, however, he believed them to be protozoa. They were identified as fungi in 1912 by da Rocha-Lima [3] by comparing them to Cryptococcus farciminosus, the causative agent of epizootic lymphangitis in horses. This was confirmed in 1934 by culture.[4]

H. capsulatum is a member of the fungi imperfecti.[5] It grows in two forms—a yeast phase that is pathogenic for man and is the form normally found in the tissues, and a mycelial phase produced by culture at room temperature on such media as Sabouraud's glucose agar. The mycelial form produces large chlamydospores, 15μ to 20μ in diameter, surrounded by a double refractile wall through which finger-like projections of cytoplasm extend, giving the structure a peculiar "cogwheel" appearance likened, by one observer, to the emblem of the Rotary Club.[1] These chlamydospores

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Otolaryngology (Capt. King); Internal Medicine (Capt. Cline), Walter Reed Army Hospital, Walter Reed Army Medical Center.

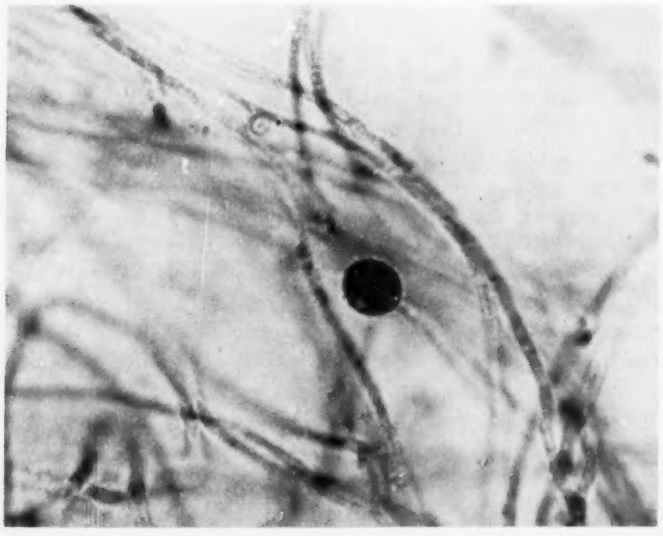

Fig. 1.—Chlamydospore of H. capsulatum from biopsy of larynx.

are specific for the histoplasmosis organism (Fig. 1).

Histoplasmosis lesions have been found in almost every organ of the body.[6-8] From indirect evidence, the lungs appear to be the main site of predilection. As the disease primarily affects the reticuloendothelial system, it is not surprising that the liver and spleen are usually widely involved. When death results, it is usually from destruction of the adrenals by the organisms.[7,9] Lesions of the nose, mouth, and pharynx are not uncommon. In 1947 Curtis and Grekin[8] reviewed one series in which four patients had involvement of the nasal mucosa, seven had tongue lesions, five had lesions of the buccal and labial mucosa, five had lesions involving the palate, three had gingival lesions, and seven had the hypopharynx involved. Since it was first described, only about 20 cases of laryngeal involvement have been reported,[1,5,7,9-16] but in several of these it may have been the primary site.[1,5,10-12,16] The lesions themselves are characterized by pseudotubercle formation and areas of necrosis with central proliferation. The granulomatous areas contain large numbers of macrophages, which usually surround necrotic centers. These macrophages contain the Histoplasma organisms. Giant cells, lymphocytes, and eosinophils are seen in varying numbers, while polymorphonuclear leukocytes are rare. Except for the presence of the intracellular organisms, the picture is not unlike that of tuberculosis.[6,8]

The first reported case of histoplasmosis occurred in a Martinique Negro and was reported from Panama.[2] Several of the cases following this were also reported from tropical areas, creating a misconception that the disease was largely limited to the tropics.[8] Not until after World War II did investigation in the United States reveal a large endemic belt in the Middle Eastern United States, with the center near the junction of the Ohio and Mississippi rivers.[17,18] Unlike tuberculosis, the disease is not thought to be spread either from man to man or from animal to man, although it is found in a large number of domestic animals in the same area where it is found to exist in man.[19]

Over most of the histoplasmosis belt the organism has been cultured from the soil, and it is believed that the usual route of entry is by inhalation of the contaminated dust particles. In some measure this may account for the preponderance of infection in males, because of their work in the fields,[1] although the reason for the marked predilection for the white race[20] cannot be explained on this basis. A number of cases of cutaneous infection[8] may also be due to direct contact, although infection in this manner is much rarer than by the respiratory route. One author proposed that a marked deficiency of minerals in the soil of the endemic area may produce a deficiency which lowers the resistance of the residents to histoplasmosis.[1]

Except for the rare cutaneous infection, histoplasmosis occurs in two forms—a common benign form in which the lungs are the organs primarily involved, and a rare fulminating form affecting practically all the tissues of the body.[20] The symptoms include fever, anemia, splenomegaly and hepatomegaly, weight loss, anorexia, nausea and vomiting, diarrhea with or without blood, cough, rhinitis, sore throat, hoarseness, and dyspnea, depending on the organs involved.[5] In the presence of so few reported lesions of the larynx, it is surprising to note hoarseness as a common, early, and sometimes the presenting symptom. Diagnosis may be made by identification of the organisms within the macrophages on the stained slide, by culture and animal inoculation, and by histoplasmin skin tests and complement-fixation tests. In the presence of histoplasmosis, the skin tests for blastomycosis and coccidioidomycosis may be positive, although less so than the histoplasmin test, but neither of the other diseases produces a positive histoplasmin test. No false positives are seen with the tuberculin skin tests.[20]

No therapy has proved effective in the treatment of histoplasmosis. Heavy metals have produced no improvement, and the antibiotics are felt to be contraindicated.[14] Stilbamidine and propamidine have afforded some relief in the treatment of laryngeal lesions, but neither has been markedly successful. Encouraging results have been reported from the use of ethyl vanillate,[13,14] but there is insufficient material available to furnish a clear picture of its efficacy. The patient presented here was treated with amphotericin B but without success.

Report of a Case

A 32-year-old white housewife was admitted to Walter Reed Army Hospital Oct. 30, 1956, with a complaint of sore throat and difficulty in speaking. She stated that her illness had begun early the same month, when a left otitis and pharyngitis developed, for which she received symptomatic therapy at her local medical installation. The pharyngitis persisted, and she was seen in the Ear, Nose and Throat Clinic at Walter Reed Army Hospital Oct. 26, with severe dysphonia and dysphagia. The pharynx was only mildly injected, but there were several petechial spots on the arytenoids and aryepiglottic folds, and the entire larynx was somewhat edematous. The white blood count at that time was 6500, with 56% polymorphonuclear leukocytes and 41% lymphocytes. The patient was given a course of oxytetracycline (Terramycin) and rechecked on Oct. 30. The larynx was essentially the same except that the petechial spots had become small ulcerations. The patient brought x-rays with her on this occasion, and a diffuse infiltrative process was noted in both lung fields. She was admitted to the hospital with a presumptive diagnosis of tuberculous laryngitis.

Past History.—In September, 1951, the patient was admitted to a tuberculosis sanatorium in Lexington, Ky., because of a suspicious x-ray taken by a mobile unit in the same town. At that time she had a slight nonproductive cough and mild exertional dyspnea. Chest examination, routine laboratory studies, and gastric washings were all negative, as was a skin test with old tuberculin 1:100. A previous chest x-ray taken in August, 1951, showed considerable calcification in the left hilus region and suggested infiltration in the midportion of both lung fields. A check film taken in November, 1951, showed no change. Her course in the sanatorium was unremarkable. Her temperature and pulse remained normal throughout her stay. She gained 27 lb. and received no drug therapy. She was discharged as improved, from the sanatorium in December, 1951, with no clinically active tuberculosis. Her chest films were destroyed the following month and the case was considered closed.

A chest x-ray was taken in Germany in March, 1955, in conjunction with treatment for a mild asthmatic episode, and diffuse bilateral infiltration was reported. She was admitted to a United States Army Hospital at Nuremburg for further study. She reported that exertional dyspnea had existed for about three years, together with some asthmatic attacks which she had treated with a spray for from 9 to 12 months. At the time of her examination she was afebrile, but rhonchi and wheezes were audible over both lung fields. Her vital capacity was normal. Routine laboratory studies were normal except for a sedimentation rate of 28 mm. in one hour. Purified protein derivative (PPD) 1 and 2 were negative. X-rays showed a diffuse reticular infiltration bilaterally with several areas of calcification. After receiving symptomatic asthmatic therapy she was discharged from the hospital with a diagnosis of pulmonary fibrosis of unknown etiology, and advised to have serial chest x-rays taken. Her next related problem brought her to Walter Reed Army Hospital in October, 1956.

Hospital Course.—On admission to this hospital, the patient had severe dysphonia and dysphagia. Examination of the oropharynx was negative, and the larynx was the same as on her previous admission. Chest auscultation revealed multiple rhonchi and wheezes, largely expiratory, over both lung fields. No other abnormalities were noted. Her temperature was 99.6 F and her pulse 84. Laboratory findings were all within normal limits except for a sedimentation rate of 30 mm. in one hour. Hemoglobin was 14.6 gm.

During the first week of hospitalization the patient's temperature varied between normal and 100 F. She was able to swallow but not without some difficulty. About a week after hospitalization she began to show a spiking fever to 103.6 F and an increasing dysphagia, which was relieved only by codeine. A laryngoscopy performed on Nov. 9 showed increased edema of the larynx, and a tracheostomy was performed. Repeated smears and cultures of the sputum, including examination for acid-fast bacilli, showed only normal oropharyngeal inhabitants. Repeated blood cultures were negative. Skin testing with PPD 1 and 2 were negative, as were histoplasmin skin tests at 1:100 concentration.

On Nov. 18, in view of the fact that no diagnosis had yet been reached and because of the patient's continued febrile course, it was decided that she be given an empirical trial of penicillin, 600,000 units, and streptomycin, 1 gm. daily. The fever

persisted despite this treatment. From a biopsy specimen taken from the larynx on Nov. 13, H. capsulatum was finally cultured on brain-heart infusion agar, on Sabouraud's, and on blood-agar medium. From a portion of the same biopsy specimen, fixed and stained, a diagnosis of laryngeal histoplasmosis was made. On the receipt of this report, antibiotic therapy was discontinued, and the administration of the experimental drug, amphotericin B, was begun. This was raised to its full dosage of 4 gm. per day over a five-day period, then continued for seven days. It produced no clinical change in the patient's course.

A nasogastric tube was passed the latter part of November, when it became impossible for the patient to swallow, and she was thereafter maintained on Trainor's solution.* Esophagoscopy revealed no lesions. Stool examination and sigmoidoscopy showed no abnormalities, nor could fungi or other enteric pathogens be cultured from the feces. A biopsy specimen taken from the liver showed a granulomatous hepatitis, but no fungi could be cultured. A bone marrow biopsy was also negative for fungi. Repeat histoplasmin skin tests at 1:100 and 1:50 concentrations remained negative. Serological tests for fungi were performed on Nov. 15 and Dec. 3. On both occasions the complement fixation test for histoplasmin in the yeast phase was 1:128, and in the mycelial phase 1:32. Blastomycosis complement-fixation tests showed a 1:16 titer in the yeast phase. A coccidioidomycosis complement-fixation test was negative.

On Dec. 16 a course of γ-globulin was begun in an attempt to increase the patient's natural resistance to infection. Over a seven-day period 280 cc. of γ-globulin was injected, but there was no significant change in her condition. Constant analgesia was required for relief of laryngeal pain. On Dec. 24 the patient suffered an attack of respiratory insufficiency manifested by the typical wheezing of bronchospasm. This was temporarily relieved by aminophylline. During the remainder of her hospital stay, however, her respiratory insufficiency became increasingly severe. She required oxygen almost continually, and her chest x-ray showed an increase in the nodular densities in both lung fields. The liver and spleen remained clinically normal in size. Over the two-week period following her first attack of respiratory insufficiency she responded less and less to oxygen and symptomatic therapy, and she finally died on Dec. 30 during an episode of apnea and cyanosis.

Pathology.—Necropsy confirmed the diagnosis of disseminated histoplasmosis. The entire lower respiratory tract was lined with a granular yellowish material, which, on tissue culture and stain, was proved to contain H. capsulatum. Focal ulcerations, large and small, were distributed over the mucosal surfaces. The epiglottis, supraglottis, endolarynx, subglottis, trachea, main-stem bronchi, and bronchioles were diffusely involved. The epiglottis had a moth-eaten appearance and a thickened, irregular surface. The vocal cords were thickened, and the right ventricular fold was perforated. Both piriform sinuses were enlarged and ulcerated (Fig. 2). The tracheostomy opening was lined with granular material similar to that lining the rest of the respiratory tree. The lungs were moderately congested and contained many minute grayish granulomata, both discrete and confluent, about the size of a millet seed. In the right upper lobe there was a fibrocaseous nodule 3 mm. in diameter, and a similar nodule 6 mm. in diameter was found in the left lower lobe. It was felt that one of these might well represent the primary lesion from which the infection had spread. Multiple cavities, 5 to 10 mm. in diameter, were present in both lungs, and the pleura and lung fissures were lined with thick gray granulomatous tissue (Fig. 3). Similar lesions were found in the liver, spleen, hilar, parabronchial, and mediastinal lymph nodes, and there was a large granulomatous lesion in the right adrenal.

A cross section of the larynx revealed a diffuse granulomatous infiltration of the mucosa with focal

Fig. 2.—Larynx, showing destruction caused by H. capsulatum.

* Trainor's solution is a high-calorie, high-protein mixture for tube feeding, similar in content to Sustagen (Mead Johnson). Widely used at Walter Reed Hospital.

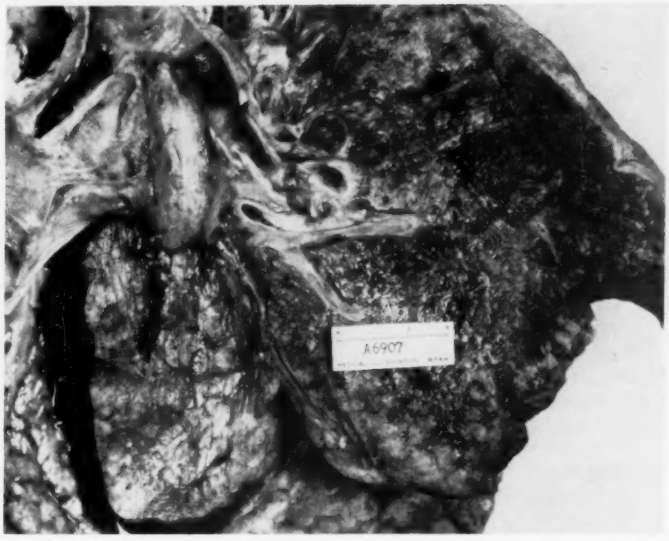

Fig. 3.—Lung, showing lesions of H. capsulatum.

erosions and shallow ulcerations. The infiltrate was predominantly composed of lymphocytes and histiocytes, many of which contained the Histoplasma organisms. The involved cells showed enlargement and some displacement of the nucleus, and the cytoplasm was entirely replaced by the small ovoid bodies of the fungi. Throughout the mucosa focal areas of necrosis were found which were rimmed by an irregular layer of lymphocytes. The disease process was primarily superficial, with only a few lesions lying deep in the underlying tissue.

Summary and Conclusions

A typical case of disseminated histoplasmosis with laryngeal involvement is described. It was possible in this case to follow the development of the laryngeal lesions from an early stage.

It is suggested that histoplasmosis may involve the larynx in a much larger number of cases than is currently reported.

No drug has yet proved its worth in the treatment of histoplasmosis. Ethyl vanillate has shown some promise. Amphotericin B, tried experimentally in the case presented, proved to be unsuccessful despite encouraging laboratory results. More study is required, however, before any definite opinion can be formed.

Histoplasmosis should be suspected when granulomatous lesions of the larynx are present, despite negative histoplasmin skin tests, and a definite diagnosis should be established by biopsy and culture.

Walter Reed Army Medical Center.

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Movements of the Superior Structures in the Laryngectomized Patient

W. H. STRUBEN, M.D., and L. van GELDER, M.D., Amsterdam

The function of the soft palate is to provide at the pharyngeal crossroad of airway and foodway a mobile separation between the nasal and the oral passage. In respiration through the nose the air can pass freely between the soft palate and the pharyngeal wall, while in swallowing the soft palate closes off this passage so that no food or drink may reach the nose.

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From the University Clinic for Ear-, Nose-, and Throat Diseases, Wilhelminagasthuis; Director: Prof. L. B. W. Jongkees.

In addition to this function the soft palate contributes to the faculty of speech as developed in the human race. The lowering of the larynx below the oral level in the evolution of biped man entails not only mouth breathing but also oral speech. (cf. Negus: "The larynx is pushed down the throat as posture became erect!") In various animals (horse, dog, etc.) in which the larynx is situated more in line with the oral cavity or even higher (in the nasopharynx), mouth breathing and consequently oral sounds are not possible. For the production of sounds these animals first

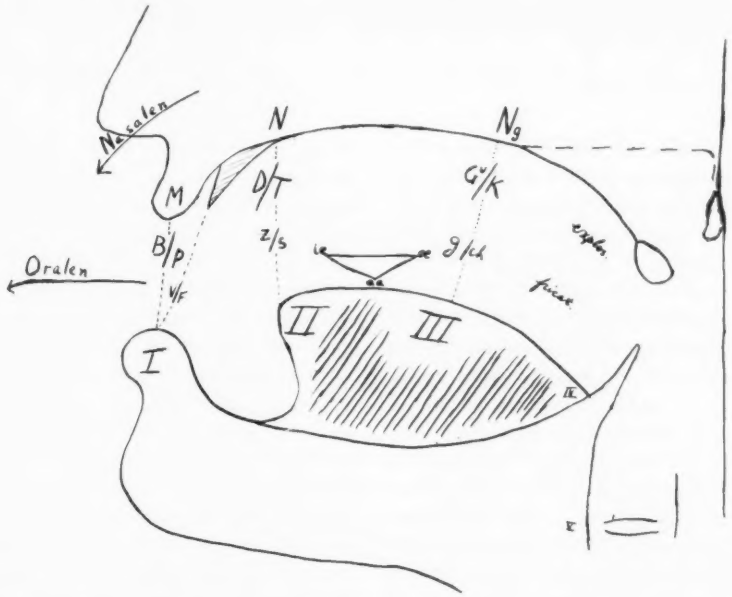

Fig. 1.—Articulation scheme showing the nasal sounds (*M*, *N*, *Ng*) and oral sounds (voiced and voiceless explosive and fricative sounds, belonging to the labial (*I*), the linguodental (*II*) and the linguovelar articulation place (*III*); the vowel triangle is represented below). The soft palate is schematically indicated; at rest and in nasals by a continuous line, in oral sounds by a dotted line.

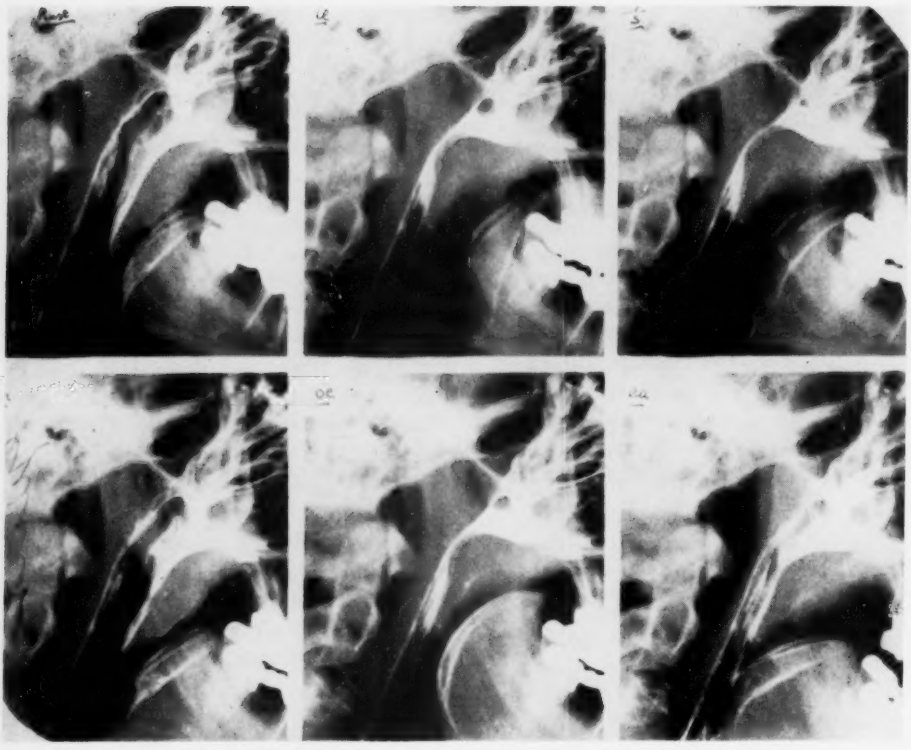

Fig. 2.—Normal speech. Position of the soft palate, from left to right: at rest; *ee*; *s*; *m*; *oo*; *ah*. Note the hooked shape of the soft palate, which touches the pharyngeal wall in oral sounds, except in *ah*.

raise their heads in order to bring the larynx below the level of the oral cavity.

If in man the nasal passage cannot be closed off by the soft palate, speech will become unintelligible because air from the lungs and larynx will escape by the nasal route without being transformed into oral sounds. Closure of the nasopharynx during speech directs the air stream from lungs and larynx towards the mouth with its resonance and articulation chamber in which the majority of sounds, after passing the larynx, are transformed into oral sounds (vowels and consonants). Only for a few so-called nasal sounds (13%) is it necessary to preserve an open nasal route, in the Dutch and English languages these sounds being *m*, *n*, and *ng*.

If by pathological causes (short palate, cleft palate, or paralysis of the palate) a leakage is present between the oral and nasal chambers, the loss of air through the nose during pronunciation of oral sounds will be serious: the oral explosive and fricative sounds are weakened and as the explosive consonants especially pass over into the correspondent nasal sounds, open nasal speech assumes an *m* and *n* character.

The diagnosis of open nasal speech can easily be made by the test of Czermak: In case of hypofunction of the soft palate dimming of the mirror appears on account of loss of air by the nose. A modern method of examining the function of the soft palate is provided by x-ray investigation with a contrasting substance or by radioscopy with the Philips image intensifier.

In 1907 Barth and Grunmach used little metal chains for visualization of the move-

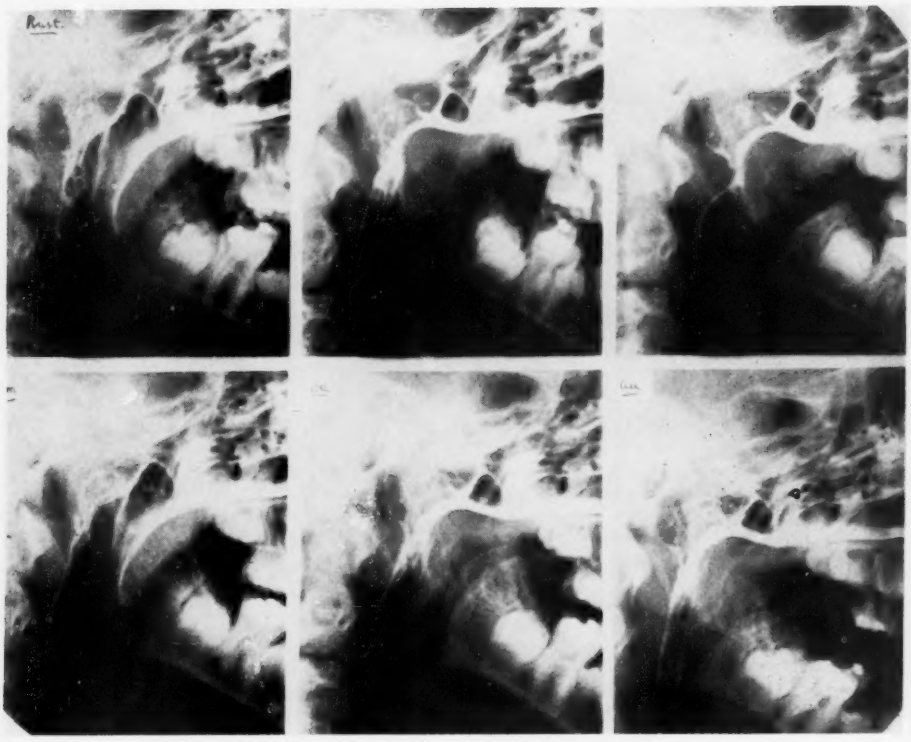

Fig. 3.—Open nasal speech with effective compensatory action: (1) by adenoid growth (see rest-position); (2) by Passavant's ridge to be formed upon the first vertebra (see *s*).

ments of the soft palate. Froeschels and Haudek (1911) used bismuth, and in 1930 Gutzmann employed barium and iodized oil (Lipiodol) to show the movements of the upper structures.

In the University Clinic of Amsterdam van Gelder has been using a method suggested by Borel and Veau from Paris. A barium suspension is introduced intranasally and intraorally. In this way illustrative roentgenograms of the soft palate and adjacent structures are obtained during the pronunciation of certain sounds. During rest (respiratory phase) the soft palate is hanging downwards along the back of the tongue. At the pronunciation of the *ee* or even more so of the *s* the soft palate is lifted and appears hooked at about two-thirds of its length. The anterior part of the soft palate is brought in line with the hard palate or even above this level and this anterior part blocks the nasal route. The posterior part with uvula has little or no significance in closing the gap between oral and nasal passage.

It will be understood that closure of the nasopharyngeal entrance to the nose is not only dependent on a well-functioning soft palate (sufficiently long and mobile) but also on the form of the nasopharynx.

If we compare the superior structures in speech of a normal person with those of a laryngectomized patient, we observe in our roentgenograms an outstanding difference is closure of the nasopharynx. In a normal person the soft palate will contact the pharyngeal wall along a distance of 1 cm. or less, whereas in a laryngectomized patient this contact with the pharyngeal wall is much more intensified and will take place along a distance of 2 to 3 cm. It is necessary for the laryngectomized patient to make

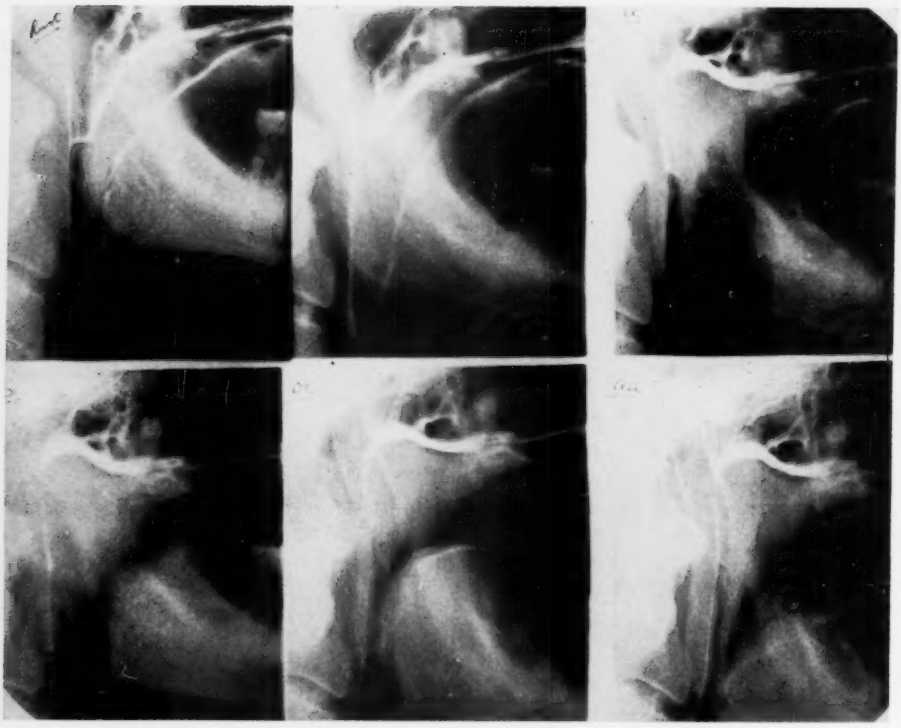

Fig. 4.—Esophageal speech in a laryngectomized patient. Note the extensive and more intensified contact between highly elevated soft palate and posterior pharyngeal wall, in an attempt not to waste any air along the nasal route during speech.

optimum use of the available volume of air collected in the upper part of the esophagus or in the pharyngeal chamber as in pharyngeal speech (Beck). An unintelligible speech of the laryngectomized patient may be the result of insufficient technique to bring air into the upper part of the esophagus (as shown by Moolenaar-Bijl and van den Berg) but may also result from insufficient closure of the nasopharynx. It is interesting, therefore, in this respect to examine the way in which nasal sounds are produced by the laryngectomized patient. Whereas in normal speech the nasopharyngeal passage is kept freely open during the pronunciation of a nasal sound (and shortly even during the preceding or following sounds, causing the so-called assimilation nasality), the nasal sound is produced in a different way by the laryngectomized patient. He will start by closing the nasopharynx, as only in this way will he be able to bring air into the upper esophagus; then after pronunciation of the purely oral sounds, the nasopharynx is opened for a very short time only to pronounce the nasal sound and immediately closed again to prevent unnecessary loss of air through assimilation nasality.

We feel that to obtain a clear voice and intelligible speech an excellent functioning of the mechanism of the upper esophagus with pseudoglottis is necessary. The same may be said of the closure of the nasal passage in pronouncing oral sounds in the laryngectomized patient. The roentgenograms accompanying this article and the roentgen film taken at the Philips Roentgen Laboratory in Eindhoven demonstrate clearly that the soft palate is able to adapt itself to the new and difficult task imposed on it

by the speech of the laryngectomized patient.

Dr. Veddema and his staff at the Philips Roentgen Laboratory, Eindhoven, assisted in the preparation of the roentgenograms.

Wilhelmina-Gasthuis.

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Tissue Reaction to Implants

WILLIAM B. BARRY, M.D., Kansas City, Mo.

Aims and Objectives

This study is presented in the hope that it will be a contribution to knowledge of tissue reaction to the commoner as well as to several uncommon materials used as implants upon the bony nasal dorsum. Dogs were used in this experiment because of their availability and because the relationships among their nasal bones, cartilages, skin, and subcutaneous tissue are reasonably similar to that found in the nasal dorsum of man.

Previous Experimental Work with Grafts in Animals and Humans

Careful search of both the dental and the medical literature reveals animal experimentation in implant materials, but nothing very closely corresponding to this experiment.

Searcy, Carmichael, and Wheelock,[8] in 1944, implanted the following substances subcutaneously in rats to determine which would be most firmly fixed with fibrous tissue: (*a*) cotton wool, (*b*) cotton string, (*c*) cotton gauze, (*d*) nylon staple (sized), (*e*) viscose rayon staple, and (*f*) ethyl methacrylate staple (sized). They found that cotton wool provoked the greatest fibrous-tissue formation and the mass was more firmly fixed in place than any of the other materials used.

Loeb,[9] in 1926, buried xiphoid cartilage with perichondrium and fat of a guinea pig in the same guinea pig (autograph) and in different guinea pigs (homograph). He removed the cartilage at intervals of from 1 day to five months and 19 days. He found that after autotransplantation, reaction in the tissues about the transplant was almost entirely lacking. After homotransplantation, lymphocytes collected about the graft, and in places lymphocytes, connective-tissue cells, and blood vessels entered necrotic portions of the cartilage. The reaction began early, reached a maximum in about three weeks, and thereafter ceased almost entirely. Both the autograph and the homograph remained up to five months and 19 days.

Peer,[1] in 1938, transplanted sections of human costal and septal cartilage preserved in 50% alcohol beneath the skin on the chest of other human beings and removed them at intervals of from seven days to 14 months. He found that a reaction of the foreign-body type was seen in the host tissues about the transplants. This reaction lasted until the 32d day and was largely absent in the sections removed later. From 32 days to and including four months the grafts remained as tolerated dead foreign bodies. In the sections buried for 14 months, fibroblasts from the host tissue grew into the cartilage graft, splitting it into two separate masses. There was definite absorption of the cartilage, and in one area calcification of the cartilage. He concluded that one could neither advocate nor discourage the clinical use of preserved cartilage grafts on the basis of his experiments, as absorption of the cartilage and replacement by fibrous tissue and new bone, if they maintain the contour of the grafts, may constitute a desirable filling substance.

Peer,[2] in 1939, found that dead cartilage grafts buried from nine and one-half months

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From the Department of Otolaryngology, University of Kansas School of Medicine, Kansas City, Kan.

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to two years showed progressive invasion by fibrous tissue and partial absorption. In contrast to these findings autogenous rib cartilage grafts showed no invasion or absorption over the same period of time. From these experiments he concludes that autogenous rib cartilage is better material for plastic repair than dead, preserved cartilage.

Peer,[3] in 1941, found that autogenous rib and septal cartilage without perichondrium after transplantation in human beings, either beneath the nasal skin or beneath the chest skin, survives as living cartilage. It maintains the same gross bulk as when first transplanted, and is not subject to absorption. He also found that only smooth segments of septal cartilage are suitable for grafting purposes. Nicks, indentations, or fractures in the cartilage will be occupied by connective tissue after transplantation, and contracture of this connective tissue may distort or split the graft.

Brunner,[7] in 1949, after removing and examining an autogenous rib implant to the nose in the human, felt that the fate of the transplanted living rib cartilage is not determined by the tendency toward degeneration, but by the resistance of the cartilage against the surrounding connective tissue, and that the cartilage covered with perichondrium is more resistant than cartilage without perichondrium.

Fulcher and Maxwell,[4] in 1943, in a review of the data available at that time about the "Tissue Reactions to Metallic Implants," concluded that, as a result of minimal tissue reaction, tantalum is the metal of choice in those cases requiring metallic implants.

Foman, Luongo, Schattner, and Turchik,[10] in 1945, concluded, as a result of clinical experimentation, that cancellous bone grafts were far superior to those of other materials.

Goldman,[6] in 1950, reported a case in which preserved cadaveric rib cartilage which had been inserted in a nose three years previously underwent metaplasia in situ with concomitant histological features typical of viable cartilage.

Peer, Walker, and Van Duyn,[11] in 1950, stated that, since both living autogenous cartilage grafts and living autogenous septal and cancellous bone grafts survive transplantation, all may be used for grafting purposes. They feel, also, that the presence or absence of perichondrium does not appear to affect the survival of cartilage grafts.

Pressman,[12] in 1952, stated: "Tantalum mesh, therefore, rolled, moulded, and cut to contour, is a most satisfactory implant and deserves further investigation."

Cottle, Quilty, and Buckingham,[13] in 1953, concluded: 1. Autogenous cancellous bone when indicated is the best graft material available. 2. Implants of autogenous cartilage from the ribs, ear, septum, and ilium, preserved human bone and cartilage, ivory, plastics, and ox cartilage, all have a place in nasal surgery and should be used.

Hilding,[14] in 1952, described a methyl methacrylate implant, which can be shaped in a dental laboratory from wax models prepared by using a wax model of the patient's face.

Gillies and Kristensen,[15] in 1950, reported the use of ox cartilage in 65 cases of nasal defects, with only 4 failures.

Sabanas et al.,[16] in 1955, in an experimental comparison of homologous bone grafts in dogs, preserved by acetone and by freezing, found both to be satisfactory with very little difference.

Sabanas et al.,[17] in 1955, in an experimental comparison of homologous bone grafts in dogs, preserved by acetone and formaldehyde and by acetone alone, found both satisfactory as implant materials. He concluded that the absence of adverse biologic effects of formaldehyde on bone is important in that exposure of ground bone to formaldehyde will kill spore-forming organisms contaminating such bone.

Johnson,[18] in 1954, implanted polyvinyl sponges into the noses of seven dogs through incisions in the forehead. In three of the seven dogs, the sponges remained in place

throughout the one-year length of the experiment. The sponges became filled with fibrous tissue and surrounded by a firm connective-tissue pseudocapsule. In the remaining four animals, the sponge became exposed after one, two and one-half, three, and four months.

Lichtor,[19] in 1954, reports on the use of an alloplastic graft made from the inorganic salts found in bone, mixed with gelatin. Formaladehyde was added to render the material sterile and resistant to dissolution by water.

Cottle et al.,[20] in 1949, reports on the satisfactory use of cancellous bone grafts in nasal repair.

Experimental Procedure

Seventeen normal adult dogs, eight females and nine males, were selected and anesthetized with pentobarbital sodium. An incision approximately 3 cm. in length was made over the midline of the bony dorsum of the nose, through the skin, the subcutaneous tissue, and the periosteum to the nasal bones. A small piece of nasal bone was removed with a gouge, and the implant was then placed in the shallow depression thus produced. The periosteum was closed over the implant with three interrupted 00 chromic absorbable surgical sutures, and the skin closed with three interrupted silk sutures. All dogs received 300,000 units of procaine penicillin G in aqueous solution daily for seven days postoperatively. Postoperative reaction in all cases was negligible.

The animals were killed at intervals of 30 days to one year and the implanted materials removed with a portion of the surrounding skin, subcutaneous tissue, periosteum, and nasal bone. Each implant with its surrounding tissue was fixed in formalin, decalcified, sectioned in the usual manner, and stained with hematoxylin and eosin. The implants consisted of the following materials, each measuring approximately 1.5×1×0.5 cm. in size: (1) homogenous nasal bone; (2) autogenous auricular cartilage; (3) bovine cartilage; (4) autogenous cancellous bone from the ilium; (5) polyethylene plastic, and (6) alloplastic calcium, gelatin, and formaldehyde mixture.

Microscopic Findings

Figure 1 is a section of a nasal dorsum, in which a small piece of preserved homogenous nasal bone was transplanted 137 days previously, which shows a number of small fragments of eosinophilic dead bone, with relatively little surrounding reaction. There are no osteocytes which stain within the bone. The fibrous tissue surrounding the bony spicules in comparable to that seen in other adjacent areas. As studied in the multiple sections from a single animal at this one interval of time, it would appear that the bone has been resorbed and fragmented to a considerable extent, although active resorption is not apparently going on at this time because of the absence of osteoclasts surrounding the bony spicules.

Figure 2 is a section of the nasal dorsum, in which a small piece of autogenous auricular fibrocartilage was transplanted 285

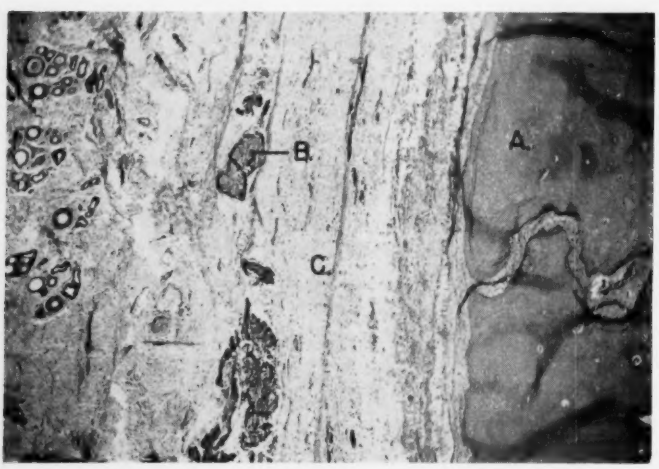

Fig. 1. — Homogenous nasal bone buried 137 days. *A*, nasal bone. *B*, small fragments of eosinophilic dead bone, with relatively little surrounding reaction. *C*, surrounding fibrous connective tissue. Reduced about 37% from mag. ×155.

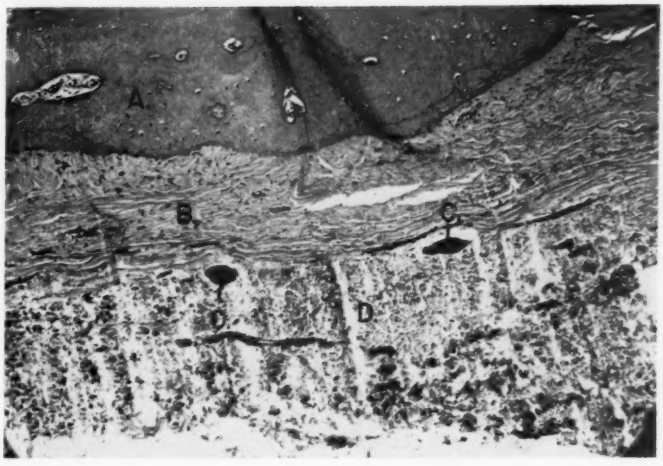

Fig. 2. — Autogenous auricular cartilage buried for 285 days. *A*, nasal bone. *B*, thickened periosteum of the nasal bone. *C*, eosinophilic debris. *D*, surrounding fibrous connective tissue. Reduced about 37% from mag. × 155.

days previously. There are several areas in which remnants of the cartilage can be readily demonstrated. In some areas this is seen as eosinophilic debris containing a few fibroblasts and ghost cells. In one area a piece of fibrocartilage is apparently intact outside of the present periosteum of the bone. In another area, there is a suggestion of bony replacement of the cartilage. Adjacent to an area with some eosinophilic bodies and fibroblasts, one can see an area within the nasal bone in which there are apparent remnants of former cartilage cells as represented by oval, eosinophilic bodies, in some instances partially surrounded by a lacunar space. Although one ought to be cautious about drawing conclusions from a single observation, it is suggestive that this represents ossification of the former cartilage.

Figure 3 is a section of the nasal dorsum, in which a small piece of ox cartilage was transplanted 191 days previously. There are several large remnants of this cartilage, which was basophilic on staining. There is active resorption of the cartilage going on, with apparent replacement by fibrous tissue rather than by bone at the site of resorption.

Figure 4 is a section of nasal dorsum, in which a small piece of polyethylene plastic was transplanted 217 days previously. The

Fig. 3. — Ox cartilage buried for 191 days. *A*, nasal bone. *B*, large remnant of ox cartilage. *C*, surrounding fibrous connective tissue. Reduced about 37% from mag. × 155.

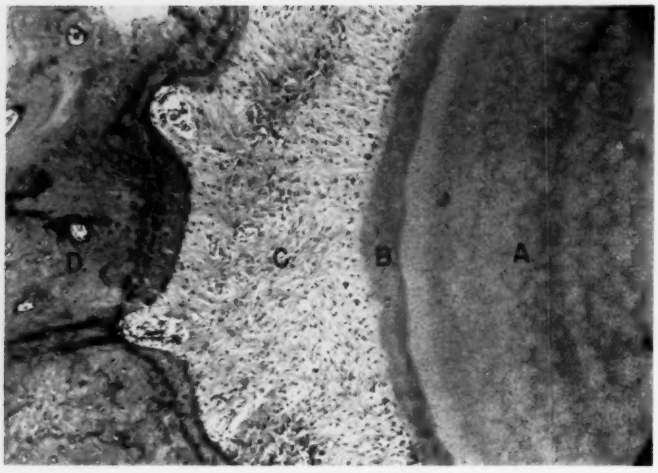

Fig. 4. — Polyethylene plastic buried for 217 days. *A*, intact plastic implant. *B*, apparent transition of collagenous fibers into the surface area of the plastic. *C*, dense band of fibrous connective tissue surrounding the implant. *D*, nasal bone. Reduced about 37% from mag. × 155.

plastic appears to be relatively intact and surrounded by a zone of dense fibrous tissue containing some inflammatory cells. It is of interest that there has been extensive proliferation of bone, so that the layer of nasal bone superficial to the plastic is now greater than the layer of bone underlying the plastic. It is not possible to say to what extent, if any, the plastic has resorbed, although it is of interest that there is an apparent transition of collagenous fibers from the dense connective tissue surrounding the plastic into the surface of the area of the plastic.

Figure 5 is a section of nasal dorsum, in which a small piece of ox cartilage was transplanted 30 days previously. This section is covered with skin beneath which numerous integuments are seen and below this there is laminated fibrous tissue in which is imbedded a large block of cartilage. In this connective tissue an occasional mass of suture material is seen. The cartilage is almost completely surrounded by a fibrous capsule, and this fibrous capsule on the under surface is abutting upon bone tissue which shows some fatty marrow spaces with osteoblastic proliferation. These marrow

Fig. 5. — Ox cartilage buried for 30 days. *A*, nasal bone. *B*, fibrous tissue. *C*, ox cartilage. Reduced about 15% from mag. × 155.

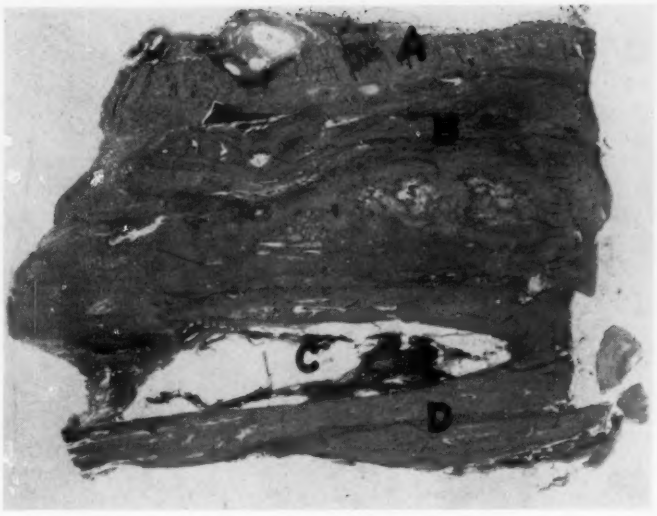

Fig. 6. — Autogenous cancellous hip bone buried for 30 days. *A*, skin. *B*, fibrous tissue. *C*, bone graft. *D*, nasal bone. Reduced about 15% from mag. × 75.

spaces are well vascularized, and the bone shows very little change of moment. Moreover, the cartilage is not significantly altered.

Figure 6 is a section of nasal dorsum, in which a small piece of autogenous cancellous bone from the ilium was transplanted 30 days previously. The bone graft is partially replaced by ingrowth of fibrous tissue, and there is a zone of fibrous tissue between the graft and the nasal bone in some areas, whereas in other areas there seems to be a connection between the graft and the nasal bone. There is definitely some absorption of the graft, however, and replacement by fibrosis, which is penetrating into the marrow spaces. At the point of contact in some areas there is some osteoblastic hyperplasia.

Figure 7 is a section of nasal dorsum, in which a small piece of alloplastic calcium mixture was transplanted 30 days previously. The section shows some bluish-purplish to reddish-staining calcific material

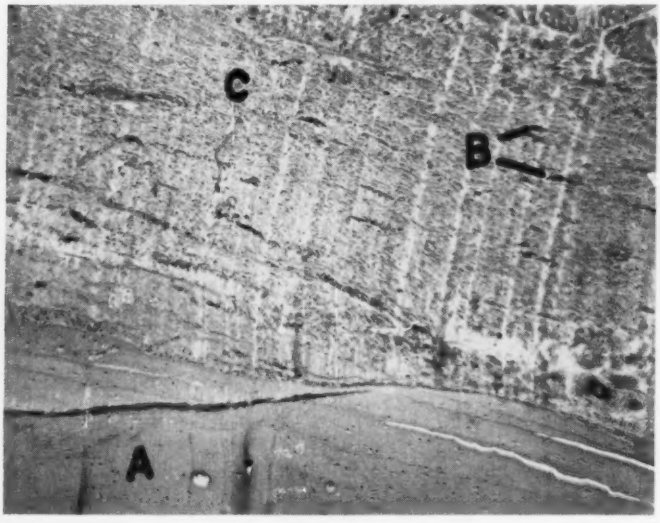

Fig. 7.—Alloplastic calcium graft buried for 30 days. *A*, nasal bone. *B*, alloplastic graft remnants. *C*, fibrous tissue. Reduced about 15% from mag. × 75.

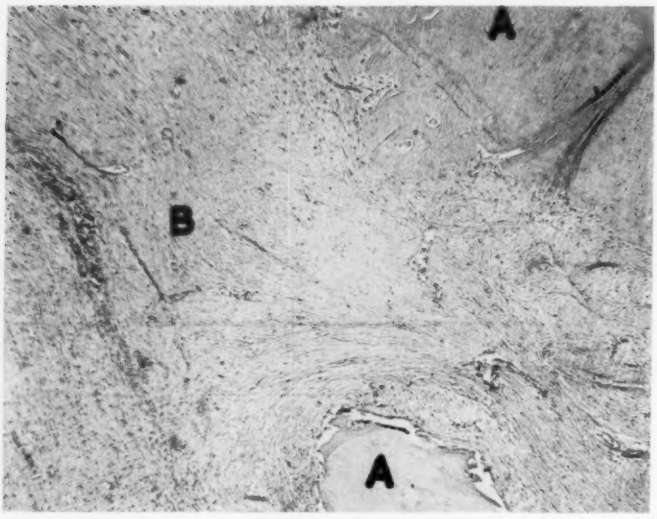

Fig. 8.—Alloplastic calcium graft buried for 105 days. *A*, nasal bone. *B*, dense fibrous tissue. Reduced about 15% from mag. × 75.

surrounded by a zone of fibrocytic cells. There are many of these deposits. The underlying bone is covered with a layer of dense laminated fibrous tisue, and the surface of this fibrous tissue is also covered with proliferating fibrocytes which are attempting to wall off the granular calcific material.

Figure 8 is a section of nasal dorsum, in which a small piece of alloplastic calcium mixture was transplanted 105 days previously. In this section, the bone is covered by thick laminated fibrous tissue containing some thick-walled blood vessels. The marrow spaces show replacement fibrosis in some areas. A few scattered lymphocytes are found in fibrous tissue overlying the bone, and none of the alloplastic material remains.

Summary and Conclusions

In summary of this work and of the literature on implants to the nasal dorsum, I feel that interesting and in some instances, definite conclusions can be drawn.

1. Peer, in his excellent work of 1938-1941, proved conclusively that autogenous rib and septal cartilage does live, and serves as a satisfactory grafting material.

2. The nasal dorsum of the dog tolerates implants of many kinds of material with only a very minimal acute inflammatory reaction.

3. Clinical experience of Cottle, Foman, and others has proved repeatedly that autogenous cancellous bone from the ilium, used as a graft to the human nasal dorsum, does firmly unite to the nasal bones and makes a very satisfactory graft for small or large defects. However, the contact surfaces must be raw, and the contact must be firmly maintained for 7 to 21 days to assure a firm union. If both of these conditions are not met, the graft will absorb.

4. In the dog, firm contact between the graft and the nasal bone is almost impossible to obtain, and therefore absorption of autogenous cancellous bone grafts occurred in three of the four dogs with such grafts. In one dog killed after 30 days, definite junction of the bony graft with the nasal bone can be demonstrated, with osteoblasts demonstrated along the raw edge of the nasal bone and on the opposing surface of the graft.

5. Implants, other than autogenous bone and cartilage, such as preserved homogenous bone and cartilage, ox cartilage, plastics, and artificial compounds, all produce a chronic inflammatory reaction of mild degree with definite proliferation of fibrous

tissue to surround, and, with the exception of the plastic material, to eventually replace the graft. In small defects, this fibrous replacement of the grafting materials may fill in the defect adequately.

6. Polyethylene plastic remained practically intact for seven months and was surrounded by a dense layer of fibrous tissue, with evidence of proliferation of the nasal bone about the graft. There was little evidence of inflammatory reaction at this time. In the four dogs in which this graft was placed, there was no extrusion.

7. The alloplastic calcium grafts were fragmented but present after 30 days, but were completely replaced after 105 days. There was a mild inflammatory reaction at 30 days and very little after 108 days, but an extensive fibrous tissue proliferation was found replacing the graft.

4600 Nichols Pkwy. (12).

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Arteria Stapedia, in Bilateral Persistence

GEORGE KELEMEN, M.D., Boston

Hyrtl, in 1836, called attention to an artery which, similarly to the vessel found in some hibernating animals, ran across the obturator foramen of the stapes. A total of nine relevant observations became known. When the following description is counted as 13—or, as the phenomenon was found to be bilateral, as 13 and 14—it can be said that in the course of 120 years a dozen plus is the number of similar findings; in other words, a decade may pass before another example is added to the list.

A 24-year-old white woman, who had had a single previous pregnancy, which, like the present one, was smooth, gave birth in the Boston Lying-in Hospital, by premature labor at 34 weeks, to a fairly well-nourished girl. The weight was 2910 gm.; CR length 30, and CH length 38.5 cm.; the cranial diameters were as follows: BP 8; SOM 10; SOF 10.

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From the Department of Otolaryngology, Harvard Medical School, and the Department of Otolaryngology, the Massachusetts Eye and Ear Infirmary. Aided by United States Public Health Service Research Grant B-1272 (C).

At autopsy a short forehead was found, and widely separated sutures; the auricles appeared to be normal. The protocol contained a long list of congenital malformations: menigoencephalocele in the occipital region; microcephaly; microgyria; hypoplasia of the brain stem; bilateral microophthalmia; multifocal cerebral calcifications; bilateral harelip, and cleft palate; persistent ostium primum; persistent left duct of Cuvier; stenotic aortic ring; cleft mitral valve; slight left ventricular hypertrophy; two accessory spleens; in the liver sclerosis and bile duct proliferation in the periportal regions; uterus didelphys; polycystic kidneys, which represented the most impressive defect, having a weight, together, of 2035 gm. (1000% of normal); bilateral clubhand and clubfoot; the left hand had six fingers, and the feet had six toes.

The temporal bones were released for further investigation. In this Dr. Kurt Benirschke, pathologist, Boston Lying-in Hospital, collaborated. His autopsy report included the malformations enumerated above. According to his opinion, the disturbances of organ formation can be assumed to have begun after the first embryonic month, extending probably to the third month, and the ultimate effects were incompatible with extrauterine life.

Fig. 1.—Tubal cartilage; stapedial artery shortly after origin from the internal carotid; deep excavation in the apical part of the cochlear capsule. Hematoxylin-eosin; reduced about ⅓ from mag. × 12.5. L. S.

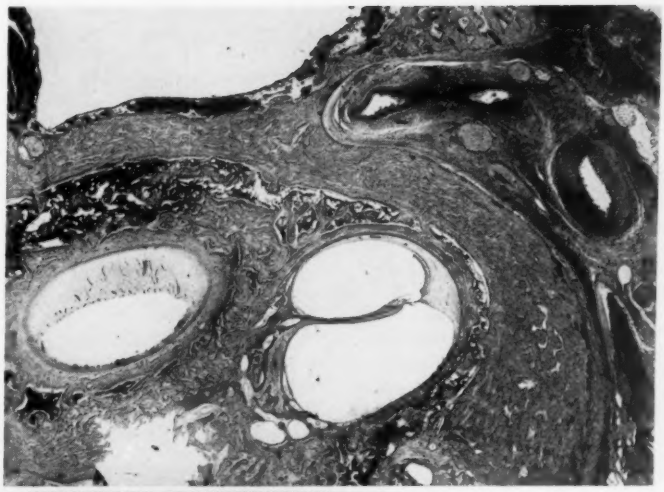

Fig. 2.—Vessel in own canal approaching the bottom of the tympanic cavity. Heidenhain-Mallory; reduced about ⅓ from mag. × 12.5. L. S.

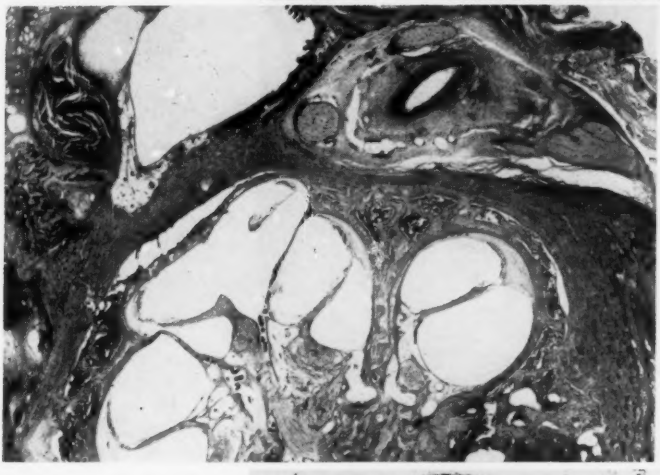

Fig. 3.—Vessel in own canal, with concomitant nerves; in its vicinity the cochlear capsule thinned out with suppression of the enchondral layer; scalae of the basal and the middle turn at obtuse angle to each other by pressure of the artery in the direction of the interscalar septum. Heidenhain-Mallory; reduced about 40% from mag. × 12.5. L. S.

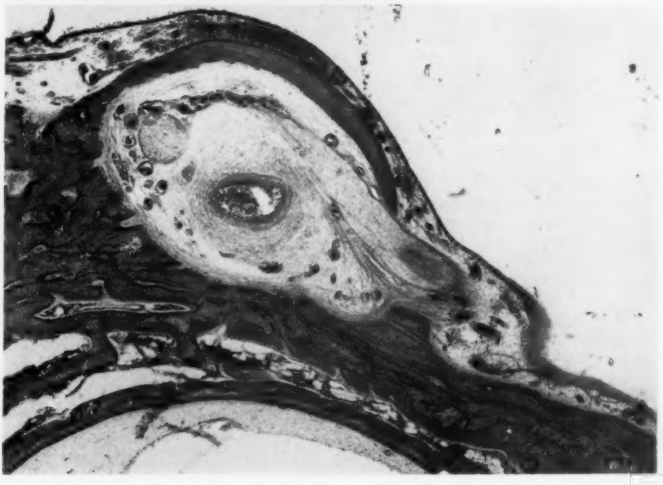

Fig. 4.—Bony canal of vessel on the promontory, in cross section, opening upward with a sharp lip. Hematoxylin-eosin; reduced about ⅓ from mag. × 35. R. S.

After the usual preparation, including embedding in celloidin, the right temporal bone was sectioned in the horizontal, and the left in the vertical, plane.

At microscopical examination both ears were found almost completely free of mesenchyme. Hyperemia of the mucous membrane, with some extravasation, was present in the middle ear on both sides. The cisterna perilymphatica contained a small amount of transudate. The cochlear aqueduct, at the left side, was dilated in its entire length. It contained some blood, and so did the niche of the round window. At the left there was engorgement in the vascular stria. As nothing remarkable was noted otherwise, the description will be restricted to the malformation itself, of identical nature on both sides. Right and left sides will be described together, while the illustrations will be chosen from the horizontal or the vertical series, respectively, according to whether clearer information is given by the one or the other.

The origin of the vessel—undoubtedly from the internal carotid—could not be traced, as it occurred below the limits of the sectioned block. When seen first in the neighborhood of the Eustachian tube (Fig. 1) it formed its canal by deep excavation of the cochlear capsule near the top of the latter. It is a vessel of considerable size, although it fills only a part of the canal. This canal becomes more proportional as it winds its way in the direction of the tympanic floor (Fig. 2); it is shown here just before drilling through the floor of the tympanic cavity and still separated from its lumen by a thin layer of bone. Nerves and small concomitant vessels also run in the canal. The depression in the wall of the cochlear capsule becomes deeper where the force

Fig. 5.—Bony canal of vessel along the promontory, in longitudinal section. (Detachment from promontorial wall artificial.) Hematoxylin-eosin; reduced about ⅓ from mag. × 35. R. S.

Fig. 6.—Cross section of the vessel between the stapedial crura. Heidenhain-Mallory; reduced about ⅓ from mag. × 35. R. S.

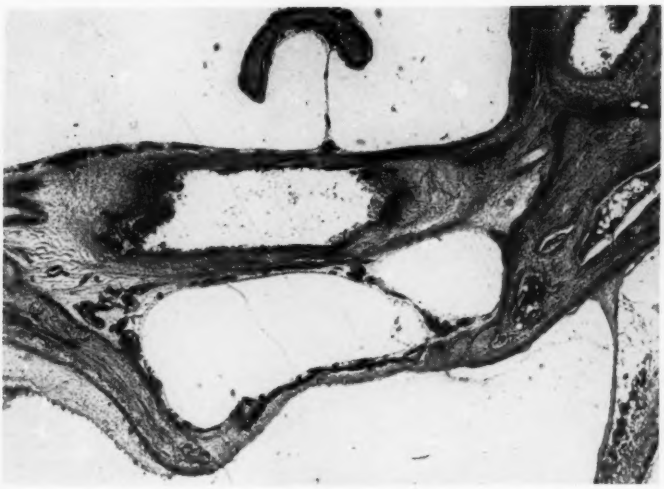

Fig. 7. — Longitudinal section of the vessel, crossing the obturator space of the stapes; connecting bands, carrying fine vessels, to the base and the neck. Heidenhain-Mallory; reduced about ⅓ from mag. × 12.5. L. S.

Fig. 8.—Vessel entering the facial canal; the nerve surrounded, from above, by the perifacial venous plexus. Heidenhain-Mallory; reduced about ⅓ from mag. × 12.5. L. S.

exerted by the pressure of the artery in the direction of the interscalar septum has caused the basal turn to deviate in an obtuse angle (Fig. 3); in the canal of Rosenthal many of the spaces are devoid of nervous elements.

After it has penetrated the floor of the middle ear (Fig. 4) the bony canal runs upward, hugging the promontorial wall, until it ends in a sharp lip. At this point, the artery is crossed by the nerve, and the nerve then lies exterior to the vessel, which continues its course along the promontory under the mucous membrane (Fig. 5, in which the detachment of the vessel from the osseous wall is an artificial ablation which occurred either at the time of the fixation or of the sectioning). Next the vessel is seen in cross section (Fig. 6) between the crura of the stapes and attached to the posterior crus. In longitudinal section (Fig. 7) it is shown running midway between the base and the neck of the stapes, and connected by fine bands with the base and with the neck respectively. Now the facial canal is entered (Fig. 8) through a wide opening while the elements of the perifacial venous plexus surround the nerve from above.

The exit from the facial canal could not be observed as the limit of the block was reached. The widely dilated cochlear aqueduct is shown (Fig. 9) with the stapedial artery ascending on the promontorial wall in the direction of the oval window.

In the bottom of the acoustic meatus (Fig. 10), in the region of the cribriform lamina, irregular exostoses represented further malformation.

Fig. 9.—Cochlear aqueduct, dilated along its entire course; vessel along the promontory before crossing through the stapedial space. Hematoxylin-eosin; reduced about ⅓ from mag. × 12.5. L. S.

Fig. 10.—Exostoses at the bottom of the internal acoustic meatus. Heidenhain-Mallory; reduced about 40% from mag. × 12.5. L. S.

Fig. 11.—Stillborn, female. Mesenchymal vessel, persisting in right angle to the course of the stapedial artery. Hematoxylin-eosin; reduced about 40% from mag. × 80.

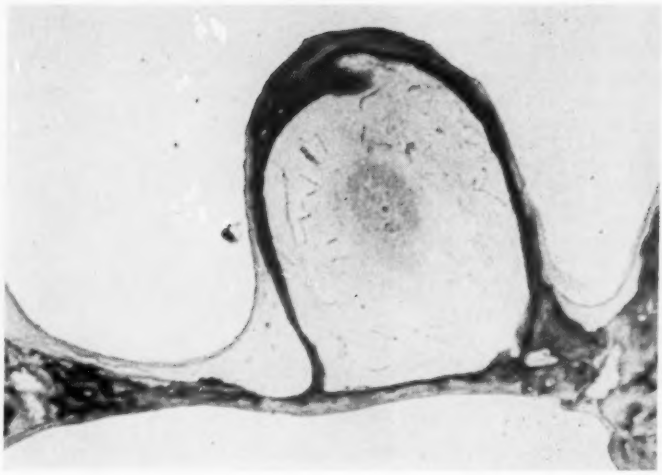

Fig. 12.—Malformation of stapes, 10-year-old girl. Fibrous plate occludes the obturator space, with dense cellular mass in the center, possibly a vestige of the stapedial artery. Hematoxylin-eosin; reduced about 40% from mag. ca. × 35.

Comment

The irregular stapedial artery is not the only vessel occurring within the intercrural space of the stapes. Mesenchyme, with ultimate remnants localized in this region, disappears in two ways: either it becomes void of vascularization before its single islands are resorbed; or the vascular skeleton remains as the last vestige before the definite clearing of the obturator space. Direction, multiplicity, and size of these vessels, as shown in an example taken from a stillborn infant (Fig. 11), preclude its confusion with a stapedial artery. Nager and Nager (1953) emphasized that none of the arteries in the stapedial region can be definitely identified with the stapedial artery of embryonic life.

In a case of aural malformation in a 10-year-old girl (Kelemen, 1943) the stapes (Fig. 12) was without connection with the single vestige of bone representing the two other ossicles, and its intercrural space was filled by connective tissue. In the center of the obturator space an oval, sharply circumscribed cellular accumulation might be identified with a persistent but obliterated vessel. A condensation ring in the

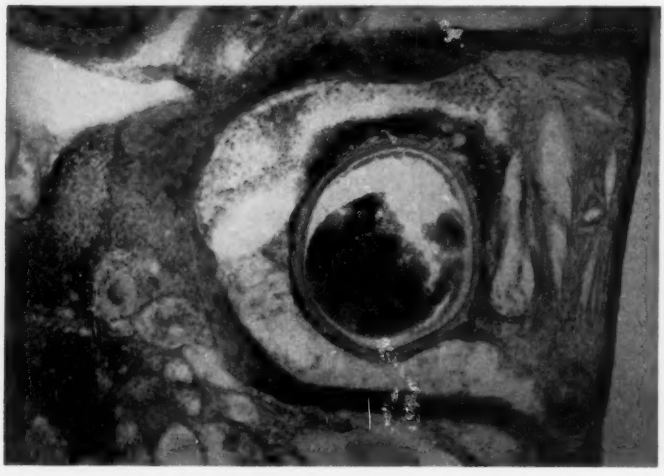

Fig. 13.—Rat, arsenic poisoning. Stapedial artery as found normally throughout lifetime (here among stapedial and peristapedial products of organization). Hematoxylin-eosin; reduced about 40% from mag. × 50.

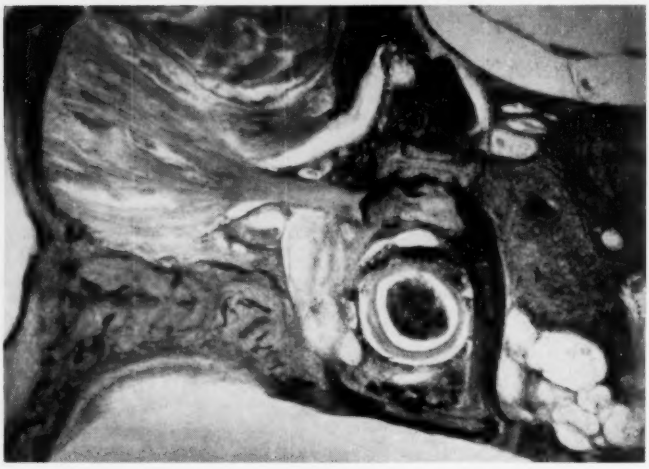

Fig. 14.—Rat, arsenic poisoning. Stapedial artery in normal persistence, in relation to tendon of stapedius muscle (among stapedial and peristapedial products of organization and superimposed recent suppuration). Hematoxylin-eosin; reduced about 40% from mag. × 50.

center of the mesenchymal mass filling the stapedial obturator foramen (Fig. 15 taken from a term-newborn boy who lived for two hours) may be interpreted as framing the former site of a stapedial artery obliterated in the course of normal embryonic evolution.

Altmann and Hagan (1948) indicated a small artery originating from the superficial petrosal branch of the middle meningeal artery, which runs down along the facial nerve, leaves the facial canal to descend farther and to pass through the crura to the promontorial arterial plexus. It can be said that its course is opposite to that of the stapedial artery.

It is not easy to construct a definite picture on the basis of the data given in the pertinent literature on the vascular conditions around this region. Tandler (1898) studied the question in great detail. Several mammalian orders show great variation in the development of the embryonic stapedial artery, from almost complete disappearance to full persistence throughout life. The constantly persistent artery crosses, as a very considerable vessel, the center of the obturator space (Fig. 12, with organization

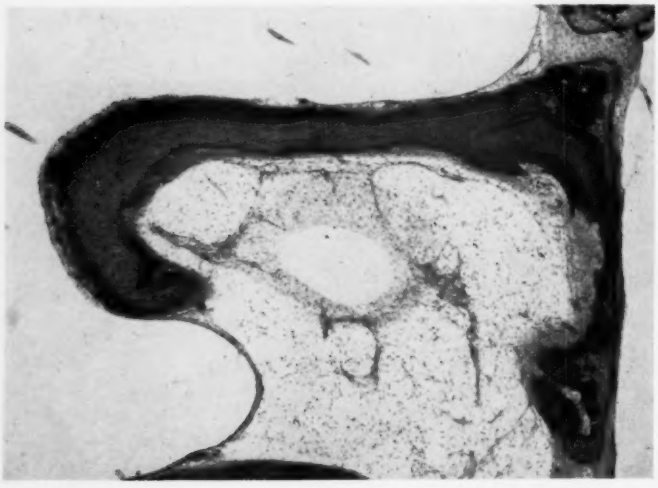

Fig. 15.—Term-newborn infant, 2 hours old. Obturator foramen filled by mesenchyme with central condensation ring corresponding to the site of the obliterated stapedial artery. Hematoxylin-eosin; reduced about 40% from mag. × 250.

products in and around the stapes; and Fig. 13, showing the vessel in relation to the stapedius muscle). Tandler explained that man, like other mammals, shows the vessel in his early embryonic period with complete disappearance later. Bast and Anson (1949) saw a rudimentary stapedial artery still present in the 25 mm. embryo. According to Broman (1899) the vessel is seen up to the end of the third embryonic month, after which a fibrous band runs through the stapes on the site of the previous vessel; in the embryo of 180 mm. any vestige of the artery had disappeared completely. Nabeya is quoted by Adachi (1927) with a contrasting view: He saw in the Japanese, newborn and adult, a very thin artery crossing the stapes, not in the center but near the base. Hyrtl (1845) and Zuckerkandl (1873) likewise mentioned this constant fine artery.

Fisher (1913) traced the development of the stapedial artery, and Altmann (1947) described in detail its origin and course; he later (1957) observed a unilateral and a bilateral persistence.

The typical form of the mammalian stapes evolves, according to Brock (1922), in strict relation with this vessel as the blastema of the stapes is shaped around it.

The monograph of Adachi gives, in numerous illustrations, the best documentation regarding the macroscopic, topical relations. The latest discussion regarding the relations of the embryonic stapedial artery to the small arteries observed in the stapedial region of the adult is by Nager and Nager.

As the internal carotid does not attain its full size when the stapedial artery remains unobliterated, malformations, atrophy or outright absence of the carotid are of significance in relation to the present topic. These conditions were studied by Wolff (1944), who gave a critical review of the pertinent literature.

Tandler (1902) mentioned that since his first publication Denker and Van Bemmelen had become interested in the question. Gradenigo (1887) was corroborated in his statements by Broman; he found in cat embryos that the stapedial artery branched off from the internal carotid, together with the primitive hyoid artery, in a hyostapedial trunk; the primitive hyoid artery soon disappears, while the stapedial artery persists until the third embryonic month.

Within the temporal bone other vascular anomalies occur more frequently. Contrary to the extreme rarity of the stapedial artery, the wide range of variations around the internal auditory artery as seen by various observers was confirmed by Nager (1954).

Found uni- or bilaterally, the course of the artery was in all observations identical, with small deviations. The vessel was found with or without further developmental abnormalities in the organism. In the case of Alexander (1899) it was observed in a bilaterally malformed fetus: Syncephalus janiceps asymmetros.

In the present case several skeletal abnormalities could be listed within the temporal bone. At the left side the facial canal presented a considerable dehiscence. The bony canal in which the artery ascends along the promontory had been seen in almost all hitherto reported cases. Here it ended in a sharp upward-pointing edge, perhaps somewhat lower than in the other instances, and left a section of the artery to run free, merely covered by the engorged promontorial mucous membrane before it entered the niche of the oval window. The cochlear capsule was adequately developed; in the apical region, however, where the pressure of the abnormal artery produced a deep trough, the capsule was thinned out at the expense of the enchondral layer (Fig. 3). Formation of a depression similar to that described here has not been observed previously.

Another abnormal feature is represented by exostoses at the bottom of the internal acoustic meatus, leaving the latter a very irregular space where the nerves have to twist their way along tortuous channels.

The almost complete lack of mesenchyme becomes the more conspicuous as it occurs here in a premature infant. The mucous membrane, inclusive of the section covering the ascending stapedial artery, shows engorgement, which may be a sign of recent differentiation from the mesenchyme.

There is no way to confirm the presence of the artery in vivo; on the other hand, its persistence does not seem to involve pathological consequences. Its discovery must remain purely accidental, although one may surmise that it is present more frequently than is revealed by the few reports on its survival. Clinically its significance might be twofold. First, as the artery is of considerable size—half the ordinary size of the internal carotid—it can be assumed that in some instances it might become a source of embarrassment in stapedial or peristapedial surgical interventions. Second, winding its way around the cochlea, it may be a source of tinnitus. This will be of a pulsating type, and, if inference is possible on the basis of the present report, of low frequency as it approaches the apical turn, even scooping out the cochlear capsule in this region.

It may be of interest to quote the remarks of Hyrtl as contained in his communication of 1845. He took it for granted that the pulse of the artery where it persists in mammals, free or enclosed by a bony canal, will produce vibrations of the stapes resulting in a continuous noise; but this noise does not cause any disurbance, as the animals are used to it; their auditory perceptions will remain undisturbed, as the miller is not troubled by the clapping of his machinery; the noise may even be useful in keeping these shy animals in a continuous state of alarm. But already, reporting his first observation in 1836, Hyrtl postulated the presence of this artery as cause of habitual tinnitus; no more can be added, even today, as to a possible symptomatology in connection with this rare phenomenon of persistence.

Finally, it might be stated that an accumulation of reports on this condition hardly can be expected; routine examinations of temporal bones by serial sectioning become, if anything, less frequent than in the past. On the other hand, the artery is of a size that cannot be overlooked, and one has to remember that the first observations on its existence were gained at macroscopical dissections, without the control of histology. More emphasis should be given, in this connection, to developmental abnormalities of the internal carotid. To the few competent reports along this line more would be added except for the reason that these conditions, at routine autopsy, easily escape attention.

Summary

Bilateral persistence of the stapedial artery is described in a premature newborn girl as one among a multitude of congenital malformations. Since the first report was given, by Hyrtl in 1836, this is the 10th and the 11th observation respectively.

The abnormal vessel originated from the internal carotid, broke through the bottom of the tympanic cavity, ran upward in a bony canal along the promontory, crossed the obturator space of the stapes, and entered the facial canal. Its probable ramification on the dural surface could not be followed because of the limitations of the specimen. This course appears to be practically identical in every case described hitherto.

A short survey of the extant reports is given and the possible clinical implications are discussed.

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Hearing Rehabilitation Centers in the United States

MARION P. DOWNS, M.A., Denver, and EVA T. CARSON, A.B., New Haven, Conn.

This comprehensive listing of the audiology and hearing rehabilitation centers in the United States is made available to the otolaryngologist so that he may refer his patients for competent hearing testing and educational rehabilitation. It also serves to inform the otolaryngologist of the status of the Centers' personnel in relation to the professional organization which establishes standards for audiologists, namely the American Speech and Hearing Association. This association certifies audiologists who have completed certain required course work and professional training. Two levels of certification are granted, Basic Hearing Certification and Advanced Hearing Certification, designated in the listing as BH and AH respectively. The Association also offers two levels of certification in Speech Correction, Basic Speech (BS) and Advanced Speech (AS).

The hearing certification requirements of the ASHA are listed below, and will furnish a guide as to the competency of audiologic personnel, whether certified by the ASHA or not.[1]

Requirements for Basic and Advanced Certificates in Hearing

1. Basic areas: Semester hours: 6 for BH, 9-12 for AH
   Phonetics, anatomy and physiology of the vocal mechanism, anatomy and physiology of the ear, speech and voice science, experimental phonetics, psychology of speech, acoustics, physics of sound, etc.

2. Specialized, professional course content in audiology: Semester hours: 12 for BH, 21-24 for AH
   * Hearing problems and hearing testing
   * Speech reading
   * Auditory training

   Elective: Hearing aids, hearing clinic management, aural rehabilitation, audiologic instrumentation, speech for the aurally handicapped, field work in audiology, seminar in audiology, preschool deaf child, education of the deaf, etc.

3. Specialized, professional course content in speech correction: Semester hours: 3 for BH, 6-9 for AH
   * Speech pathology

   Elective: Stuttering, voice disorders, articulation disorders, cleft palate, cerebral palsy, aphasia, etc.

Submitted for publication Dec. 19, 1957.

University of Denver (Mrs. Downs); Audiology Foundation, 33 Trumbull St., New Haven, Conn. (Mrs. Carson).

4. Other areas: Semester hours: 9 for BH, 15-18 for AH
   * Child psychology (or child development)
   * Psychology of adjustment (or mental hygiene)
   * Candidates for the Advanced Certificate must present a total of 12 semester hours in psychology beyond the first course.

   Electives in appropriate areas

   Total semester hours required: 30 for BH, 60 for AH

5. Clinical practicum:
   * Basic: at least 200 clock hours; Advanced: additional equivalent of 3 semester hours (135 additional clock hours) in advanced practicum (up to 20 hours of speech practicum may be applied).

6. Professional experience:
   * Basic certificate: one year of preregistered experience following the completion of the above listed academic requirements and clinical practicum.
   * Advanced certificate: four years of preregistered experience following the completion of the above listed academic and clinical practicum requirements for the basic certificate.

7. Recommendation:

   For Advanced Certification the applicant shall present a favorable recommendation from the director of the speech and hearing program in a department or institution offering advanced degrees in the field of hearing and speech.

8. Examination:

   For Advanced Certification the applicant shall pass both sections of a written and oral examination. Other suitable evidence, as determined by the Committee on Clinical Standards in Hearing, may be accepted in lieu of the examination upon Council approval.

* Content of these items is required.

The following listing of hearing centers was compiled from questionnaires sent to the centers, and returned during the period of Aug. 1, 1957, to Nov. 15, 1957.

When questionnaires were not returned, the information from the previous survey was used,[4] and marked with an asterisk. Thirteen new centers were added to this year's listing, and six formerly listed centers were dropped. Exclusion was made on the basis of inadequate equipment, dissolution of the Center, or failure to reply to questionnaires since 1955.

The sponsorship of the 152 centers can be broken down as follows:

Universities	48
Medical schools or hospitals	43
Hearing societies	26
Federal agencies	12
State agencies	10
Public agencies	8
Private	5

Listing in this survey does not constitute endorsement by any organization for the services rendered by the center. Any changes or corrections in listing should be sent to The Audiology Foundation, 175 St. Ronan St., New Haven 11, Conn.

Number of Center	Medical Supervision: Oto. C	Oto. S	Phys. C	Phys. S	Referrals: Med. Ex.	Med. P	Med. O	Restrictions	Total Number	Personnel: Degrees B.A.	B.S.	M.A.	M.S.	Ph.D.	M	F
1		x				x	x	P	5	2			1	2	5	
2			x			x	x	VP	12	6		3	1	2	10	2
3	x					x	x	P	1					1	1	
4			x		x			VP	5	1		2		1	2	
5	x				x			V	5			4		1	5	1
6	x				x			C	2			1	1		2	1
7	x					x	x	C	38	3	3	10	2	6	0	
8			x			x	x	P	4			2		2	4	
9			x			x	x	P	3			2			2	
10		x			x			V	4	1		3			2	
11	x				x			P	11	2		4		3	6	1
12			x			x	x	P	3					3	3	2
13			x			x	x	P	2					2	2	
14				x	x			C	6	1		5			6	
15			x			x	x	P	3			1		1		
16			x			x	x	VP	3			3			1	
17						x	x	P	1					1	1	
18			x			x	x	P	4	1	1				1	
19		x			x			VP	4		2	1	1		4	
20	x					x	x	VP	3			3			3	
21		x			x			V	39	8	2	9		4	12	
22	x					x	x	C	3	1	1	1			3	
23	x					x	x	P	5	1		2		2	3	
24						x	x	P	2		1	1			2	
25						x	x	V	10	3		3		3	9	
26	x					x	x	P	8		2	2			2	
27			x			x	x	P	6				4	2	6	2
28				x	x			V	2			2			2	
29			x			x	x	P	6			1			1	
30				x		x	x	V	2			2			2	
31	x					x	x	P	15	4	4	1		1	5	2
32			x			x	x	P	21	11		8		1	12	
33			x			x	x	P	17		5		6	6	12	
34			x			x	x	P	6	2		2		2	4	1
35				x		x	x	V	6			6			6	
36	x					x	x	P	1	1					1	
37		x				x	x	C	2	1		1			2	
38	x					x	x	P	7			3			1	
39		x				x	x	P	6			4	1	1	6	1
40		x				x	x	P	1				1		1	
41	x		x			x	x	P	8	1	2	4		1	3	
42		x				x	x	P	4	1		2			3	
43				x		x	x	P	6			3		2	2	1
44	x					x	x	P	12	1	3	4	2	2	8	1
45	x					x	x	P	8		2	5		1	4	1
46	x		x			x	x	P	1					1	1	
47			x			x	x	P	3					1	3	
48				x		x	x	P	7				3	4	7	1
49		x			x			VP	6			4		2	6	
50	x					x	x	P	20	5		5	5	4	14	2
51			x			x	x	P	1				1			
52			x			x	x	P	1	1						
53				x		x	x	P	2					2	2	
54	x					x	x	P	5	2		1	1		2	
55						x	x	P	1			1			1	
56	x					x	x	P	9			2		7	9	4
57		x			x			VP	7	1		4		2	4	
58		x				x	x	P	6				3	3	4	
59	x					x	x	VP	22	10		9		3	12	1
60	x				x			VP	8	7	1	4		1	3	
61	x					x	x	P	7	2		2	1	2	4	
62	x					x	x	P	2	1			1		1	
63						x	x	P	3			3			1	
64		x				x	x	VP	6			3		3	4	2
65		x			x			P	3					1		
66			x			x	x	P	10	1		4	1		6	
67	x					x	x	P	7		3	2		2	6	1
68				x	x			V	3			3			2	
69			x			x	x	P	7	2		5			4	
70		x				x	x	P	7	2	3	1		1	1	
71		x				x	x	P	3		1		1	1	2	
72			x			x	x	P	5	2	1	1				
73	x					x	x	P	10	3		5		1	5	
74			x			x	x	P	10			5		4	8	2
75		x				x	x	P	3	1				2	3	
76	x					x	x	VP	5	1		3		1	3	1
77						x	x	P	3			1		2	2	
78						x	x	C	4			4			1	
79	x					x	x	P	1			1				
80	x					x	x	P	2				2		2	
81	x			x		x	x	C	20	4		6			6	
82			x			x	x	P	5			4				
83						x	x	P	4					4	4	1
84		x			x			VP	3	1		1		1	2	

Certification				Equipment						Services							
Audiology and Speech																	
AH	BH	AS	BS	Pure-Tone Aud.	Speech Aud.	Sound S	Rooms D	PGSR	SPECIAL	Pure-Tone Audio.	Speech Audio.	H/A Selec.	H/A Demon.	Lipreading	Auditory Train.	Speech Train.	Psych. Testing	Counsel. & Guid.
1		1	1	3	1	1	1	x		x	x	x		x	x	x	x	x
1		2	1	4	1	2	1	x		x	x	x	x	x	x	x	x	x
	1		1	1	1		1			x	x	x	x	x	x	x	x	x
				2	1		1	x	x	x	x	x	x	x	x	x	x	x
			1	5	2	2	2	x	x	x	x	x	x	x	x	x	x	x
1			1	2	2	1	1	x		x	x	x	x	x	x	x		
				4	2		1	x	x	x			x	x	x	x	x	x
	1	2	1	5	1	2	1	x	x	x	x			x	x	x		
				2	1		1			x	x	x		x	x	x	x	x
	1		1	5	2	1	2	x	x	x	x	x	x	x	x	x	x	x
1	1	1	1	4	3		2	x	x	x	x	x		x	x	x		x
1		1		4	1	1	1	x		x	x	x	x	x	x	x		x
1				1	1		1			x	x	x		x	x	x	x	x
		1	3	1		1				x				x	x	x	x	x
				1	1	1				x	x			x	x	x		x
	1			4	2		2	x		x	x	x		x	x	x	x	
				1	1	1			x	x	x			x	x	x	x	x
				2	1	2				x	x		x	x	x	x		x
				7	4	3	1	x	x	x	x	x		x	x	x	x	x
	1			3	1	2	1	x		x	x	x	x	x	x	x	x	x
1	2		4	14	5	5	4	x	x	x	x	x	x	x	x	x	x	x
				2	1	1		x	x	x	x	x	x	x	x	x	x	x
			2	3	2	2		x		x	x	x	x	x	x	x	x	x
				1	1	1				x	x	x		x	x	x		x
	3		3	4	3	1	2	x	x	x	x	x		x	x	x		x
1		1		2	1	1		x		x	x	x	x	x	x	x		x
		1	5	4	1	2	1	x		x	x	x	x	x	x	x	x	x
		1		3	2	1	2	x	x	x	x	x	x	x	x	x		x
				1		1				x				x	x	x		x
	1			4	2	1	2	x	x	x	x	x	x	x	x	x	x	x
		2		1		1				x				x	x	x	x	x
1	1	1	2	6	1		1			x	x	x	x	x	x	x	x	x
		2	4	8	1	2	1	x		x	x	x	x	x	x	x	x	x
1	2		1	7	1	5	2	x	x	x	x	x		x	x	x	x	x
			1	9	3	2	3	x	x	x	x	x		x	x	x	x	x
				1	1	1				x	x		x	x	x			x
1			1	3	1		1		x	x	x	x	x	x	x	x	x	x
				1	1	1		x		x	x	x					x	x
1		1	2	4	3	2	3	x	x	x	x	x		x	x	x	x	x
	1		1	2	1	1	1			x	x	x	x	x	x	x	x	x
	1		1	4	2	1	2	x		x	x	x	x			x	x	x
1	1	1		2	1	1	2	x	x	x	x	x	x	x	x	x	x	x
		1		6	4	1	2	x		x	x	x	x	x	x	x	x	x
1	2	1		10	3	1	2	x	x	x	x	x		x	x	x	x	x
2		2		4	1	1	1	x	x	x	x	x		x	x	x	x	x
1				8	1	1	1	x		x	x	x		x	x	x	x	x
		1	1	1	2	1				x				x	x	x	x	x
	1	2		7	1		1			x	x	x	x	x	x	x	x	x
2	1		1	8	3		3	x	x	x	x	x	x	x	x	x	x	x
2		4	4	11	2	4	2	x	x	x	x	x	x	x	x	x	x	x
				4	1	2				x	x	x	x	x	x	x	x	x
				5	1	10	1		x	x	x	x	x	x	x	x	x	x
		1	1	4	1	1				x	x		x	x	x	x	x	x
	1			3	1		1	x		x	x	x	x	x	x	x	x	x
		1		1		1				x				x	x	x		
1		4	3	4	1	3	1	x		x				x	x	x	x	x
	1	1		4	2	3	2	x	x	x	x	x	x	x	x	x	x	x
1				5	3	2	2	x	x	x	x	x	x	x	x	x	x	x
1		6	3	3	1	1	1	x	x	x	x	x	x	x	x	x	x	x
	1			5	3	1	2	x	x	x	x	x		x	x	x	x	x
	1	1	2	3	2		2	x		x	x	x		x	x	x	x	
				3	1	1		x		x	x	x	x	x	x	x		x
			1	2		2				x				x	x	x		x
		1	1	7	3	5	1	x	x	x	x	x	x	x	x	x	x	x
				3	1	1	1	x		x	x	x	x	x	x	x	x	x
				3	2	2	1			x	x	x	x	x	x	x		x
		1		2	1	1	1	x		x	x	x		x	x	x		
				7	4	1	3	x	x	x	x	x	x	x	x	x	x	x
	1	2	1	2	1	1	2	x		x	x			x	x	x	x	x
	1		1	6	1	2	2	x		x	x	x	x	x	x	x	x	x
			1	5	2	2	2	x	x	x	x		x				x	x
				2	1	1				x	x	x	x	x	x	x	x	x
	1		1	3	1		1			x	x	x	x	x	x	x	x	x
	1	4	3	5	3	3	1	x		x	x	x	x	x	x	x	x	x
1	2			4	1	1	1	x	x	x	x	x		x	x	x	x	x
1				5	3	2	2	x		x	x	x		x	x	x		x
			1	4	1	3	1			x	x	x	x	x	x	x	x	x
	1			1	1	1	1	x	x	x	x	x	x	x	x	x	x	x
				1	1		1			x	x			x	x	x		x
1				3	2		1	x	x	x	x	x	x	x	x	x	x	x
				51	4		4			x	x	x	x	x	x	x		x
				2	1	2				x	x		x	x	x	x		x
	1	2	1	4	2		1			x	x	x	x	x	x	x	x	x
1				5	1	2	1	x		x	x	x	x	x	x	x	x	x

Number of Cente	Medical Supervision: Oto. C	Oto. S	Phys. C	Phys. S	Referrals: Med. Ex.	Med. P	Med. O	Restrictions	Total Number	Personnel: Degrees B.A.	B.S.	M.A.	M.S.	Ph.D.	M	F
85			x			x	x	P	6			2		4	5	1
86		x				x	x	P	2					1	1	4
87			x			x	x	P	5	1		3		1	5	1
88				x	x			V	1				1		1	
89	x					x	x	VP	21	1	9	5	3	2	11	1
90	x					x	x	VP	5			3		2	5	
91		x				x	x	P	3	2		1			2	
92	x					x	x	P	6				1	1	4	
93	x					x	x	VP	1	1					1	
94	x				x			VP	3			2	1		3	
95		x			x			C	5				5		3	
96	x					x	x	P	9	4		2			6	
97		x			x			V	9			7			3	
98		x			x			P	8	2		3		1	7	
99		x				x	x	P	19	2		7			6	
100	x					x	x	P	4			4			4	
101	x					x	x	P	5	2		2		1	1	
102	x					x	x	P	6							
103						x	x	P	6	2		1		3	4	1
104		x			x			P	5			2		2	4	
105	x					x	x	P	12	3		8			4	
106	x					x	x	P	10	3		5		2	10	2
107				x		x	x	P	3			1			1	
108	x					x	x	VP	7	2		2		1	5	
109	x					x	x	P	4					4	3	1
110		x			x			P	3			3				
111		x				x	x	VP	1					1	1	
112			x			x	x	P	13	4		4		5	12	1
113						x	x	P	7	2	2		1	2	6	
114	x					x	x	VP	9	2		4	1	1	5	
115	x					x	x	VP	39	13		16	1	6	22	
116	x					x	x	P	4	1			2	1	3	
117				x		x	x	P	5		2	3				
118	x					x	x	VP	3			1		2	3	
119	x					x	x	P	7			6		1	6	
120			x			x	x	P	3			2			1	
121	x					x	x	VP	3			3			3	
122			x			x	x	VP	13	6		5		2	1	
123	x					x	x	VP	6		3	1	1		5	
124	x					x	x	C	15			13		1	8	
125	x					x	x	P	4			1	2			
126		x			x			P	8			3		2	4	
127						x	x	C	1					1	1	
128		x			x			P	4			1		1	2	
129		x			x			V	6	1	3	1	1		3	
130			x			x	x	P	7			5			5	
131	SEE #124															
132	x					x	x	VP	10		3		5	2	10	1
133	x				x			P	4							
134	SEE #124															
135			x			x	x	C	3			3			3	
136			x			x	x	P	25		18		3	4	15	1
137	x					x	x	P	1			1			1	
138			x			x	x	P	2					1	1	
139	x					x	x	P	7	2		1		1	4	
140	x				x			VP	9	3		6				
141	x				x			VP	22	6	1	6	5	4	15	
142	x					x	x	VP	4			2		2	4	1
143	x				x			V	2			1		1	1	
144	x				x			P	3		2	1			3	
145	x					x	x	VP	12		1	5		1	7	
146	x					x	x	VP	5		2		2	1	3	
* 147	x		x			x	x	C	2	1		1			2	
148	x					x	x	P	3					3	3	
149		x				x	x	P	5		1	1		1	2	
150	x					x	x	VP	9	5		2			8	
151						x	x	P	2	1			1		1	
152	x					x	x	VP	2		1	1			1	
† 153		x			x			P							1	1

* No information received for 1957 listing.
† Information on No. 153 was received too late for inclusion in the regular listing.

Certification				Equipment						Services								
Audiology and Speech						Sound	Rooms											
AH	BH	AS	BS	Pure-Tone Audio.	Speech Audio.	S	D	PGSR	SPECIAL	Pure-Tone Aud.	Speech Aud.	H/A Selec.	H/A Demon.	Lipreading	Auditory Train.	Speech Train.	Psych. Testing	Counsel. & Guid.
		1	4	3	3	1				x	x			x	x	x	x	x
		1		6	4		1	x		x	x	x		x	x	x	x	x
1		1	1	5	1	1	1			x	x	x		x	x		x	x
				2	1	2	1	x	x	x	x	x	x	x	x	x	x	x
4	3	1	2	6	3	3	3	x	x	x	x	x	x	x	x	x	x	x
	1	2		5	1		1	x		x	x	x	x	x	x	x	x	x
	1	1		8	2	2	1	x	x	x	x	x	x	x	x	x	x	x
				1	1	1	1	x		x	x	x	x	x	x	x	x	x
	1		1	2	1	5	1	x	x	x	x	x	x	x	x	x		
	1			3	1	1	1	x	x	x	x		x	x	x	x	x	x
	1		1	2	1	1	1	x		x	x	x	x	x	x	x	x	x
	3		3	3	1	1	1			x	x	x	x	x	x	x	x	x
	2		2	7	4	3	3	x	x	x	x	x	x	x	x	x	x	x
	1	1	2	6	2	4	2	x	x	x	x	x	x	x	x	x	x	x
1	1		2	8	2	2	1	x		x	x	x	x	x	x	x	x	x
		1	2	2	1	1	1			x	x	x		x	x	x	x	x
			2	4	3	1	1	x		x	x	x						x
				9	2	3	3		x	x	x	x	x	x	x	x	x	x
1		2	1	5	3	3	2	x	x	x	x	x	x	x	x	x	x	x
		1		5	2	1	2	x		x	x	x	x	x	x	x	x	x
				5	1	1	1	x		x	x	x	x	x	x	x	x	x
1		1	5	2		2				x				x	x	x		
				3	2	3	1	x		x	x	x	x	x	x	x	x	x
	1	1	1	2			1	x		x	x	x	x	x	x	x	x	x
1		2	1	13	3	4	2	x	x	x	x	x	x	x	x	x	x	x
				3	2	1	1	x	x	x	x	x	x	x	x	x	x	x
	1			3	2		1	x		x	x	x	x	x	x	x	x	x
		10		5	1	1				x				x	x	x	x	x
		3	1	5		2				x	x	x	x	x	x	x	x	x
	1			3	2		2	x	x	x	x	x	x	x	x	x	x	x
1	1	3	5	11	6	2	4	x	x	x	x	x	x	x	x	x		x
	1		1	4	2		2	x		x	x							x
				18	3	1	1			x	x							x
2	1			8	2	4	1	x	x	x	x	x	x	x	x	x	x	x
	1	1	3	4	2	2	1	x		x	x	x	x	x	x	x	x	x
				3	3		1			x	x	x	x	x	x	x		x
	1		1	2	1		1			x	x	x	x	x	x	x	x	x
1				5	3		3	x	x	x	x	x	x	x	x	x	x	x
	1			2	2		1	x		x	x	x	x	x	x	x	x	x
	2		2	23	3	5	5	x		x	x	x	x	x	x	x	x	x
				2		1				x				x	x	x	x	x
	1	1	3	4	3	2	2	x		x	x	x	x	x	x	x	x	x
				2	1		1	x		x	x	x	x	x	x	x		x
1	1			5	1	2	2	x	x	x	x	x	x	x	x	x	x	x
	1		1	6	2	4	2	x		x	x	x		x	x	x		x
	4		4	1	1		1			x	x							
3	1		1	5	3	3	2	x	x	x	x	x	x	x	x	x	x	x
				2	2	1	1			x	x	x	x				x	
	1		2	3	1		1	x	x	x	x	x	x	x	x	x		x
1	1	5	3	4	1		1	x	x	x	x	x	x	x	x	x	x	x
			1	1	1	1				x	x	x	x	x	x	x		
			1	1	1		1	x		x	x	x		x	x	x	x	
		1	1	3	1		1			x				x	x	x		x
				4		1	1	x	x	x	x	x	x	x	x	x	x	x
1	1	1	3	19	5	5	4	x	x	x	x	x	x	x	x	x	x	
		1	1	2	1	1	1	x		x	x	x	x	x	x	x		x
				3	2		1	x	x	x	x	x		x	x	x		x
1			2	3	1		1	x	x	x	x	x	x	x	x	x	x	x
2	1	1	3	8	2		2	x	x	x	x	x	x	x	x	x	x	x
1		1		2	1	2	2	x		x	x		x	x	x	x	x	x
		1		1	1	1		x		x			x	x	x	x		
		2		4			1	x		x		x	x	x	x	x	x	x
		1	1	5	1	4	1	x	x	x	x	x	x	x	x	x		
				3	2		1			x	x	x	x	x	x	x		x
			1	2	1		1	x		x	x	x	x	x	x	x		
			1	4	1		1		x	x	x	x	x	x	x	x		x
1				1	1		1											

The following is the key to abbreviations in the list and Table (x indicates that the item or service is offered).

Medical Supervision
- Oto. Otologist
  - C Consultant otologist
  - S Staff otologist
- Phys. Physician
  - C Consultant physician
  - S Staff physician

Referrals
- Med. Referrals from doctors
  - Ex. Exclusive medical referrals
  - P Partial medical referrals
  - O Other referrals from nonmedical sources

Restrictions (on clientele accepted)
- V Restricted to veterans only
- VP Veterans and general public
- P General public
- C Children only

Personnel (certification in American Speech and Hearing Association)
- M Member
- F Fellow
- AH Advanced Hearing Certification
- BH Basic Hearing Certification
- AS Advanced Speech Certification
- BS Basic Speech Certification

Equipment
- Pure-Tone Aud. Pure-tone audiometers
- Speech Aud. Speech audiometers
- Sound Rooms
  - S Single sound room
  - D Double sound room set-up
- PGSR Psychogalvanometer
- Special Other equipment, such as audio-delay unit, etc.

Services
- Pure-Tone Audio Pure-tone audiometry
- Speech Audio Speech audiometry
- H/A Selec. Testing and fitting of hearing aids for the patient
- H/A Demon. Trial of hearing aids without testing
- Lipreading Classes in lipreading training
- Auditory Train. Classes in auditory training
- Speech Train. Speech improvement therapy
- Psych. Testing Psychometric testing
- Counsel. & Guid. Counseling and guidance in problems of hearing loss and parental counseling.

Detailed information in regard to hearing rehabilitation centers listed below may be found in the Table.

Established Hearing Rehabilitation Centers

ALABAMA

1. Birmingham, Hearing and Speech Clinic, 1919 7th Ave. S.
   Auspices: Medical College of Alabama
   Administrative Head: Robert E. Roach, Ph.D., AH
   Medical Supervisor: Frank S. Moody, M.D.
2. Tuscaloosa, Speech and Hearing Rehabilitation Center, P. O. Box 1965, University of Alabama
   Auspices: University of Alabama
   Administrative Head: T. Earle Johnson, Ph.D., AH
   Medical Supervisor: University Hospital and Medical College of Alabama Staff

ARIZONA

3. Phoenix, Speech and Hearing Center, 2021 N. Central Ave.
   Auspices: Private
   Administrative Head: Norman E. Iverson, Ph.D., BH, BS
   Medical Supervisor: Otologists from Maricopa County Medical Society
4. Phoenix, Samuel Gompers Memorial Rehabilitation Center, 7211 N. Seventh St.
   Auspices: Maricopa County Society for Crippled Children and Adults, Inc.
   Administrative Head: Clayton Lorenzen, M.A.
   Medical Supervisor: Otto Utzinger, M.D.

CALIFORNIA

5. Los Angeles, Audiology and Speech Correction Clinic, 1031 S. Broadway
   Auspices: Veterans Administration
   Administrative Head: Edward W. Gibbons, M.A.
   Medical Supervisor: Harold Boyd, M.D., F

6. Los Angeles, Hearing and Speech Clinic, Children's Hospital, 4614 Sunset Blvd.
   Auspices: Children's Hospital
   Administrative Head: Jacqueline Keaster, M.S., AH
   Medical Supervisor: Victor Goodhill, M.D.

7. Los Angeles, John Tracy Clinic, 806 West Adams Blvd.
   Auspices: Private
   Administrative Head: Edgar L. Lowell, Administrator, Ph.D.
   Medical Supervisor: Consulting Board

8. Los Angeles, Speech-Hearing Clinic, University of Southern California, University Park
   Auspices: University of Southern California
   Administrative Head: William H. Perkins, Ph.D., AS; Victor P. Garwood, Ph.D., AS, BH
   Medical Supervisor: U. S. C. Student Health Service

9. San Diego, San Diego Hearing Society, 3843 Herbert St.
   Auspices: Community Chest
   Administrative Head: John Y. Miller, M.A.
   Medical Supervisor: John Feldman, M.D.

10. San Francisco, Audiology and Speech Correction Clinic, 42d Ave. and Clement St.
    Auspices: Veterans Administration
    Administrative Head: Lyman S. Barrett, B.A., BS
    Medical Supervisor: Walter P. Work, M.D.

11. San Francisco, San Francisco Hearing and Speech Center, 1609 Scott St.
    Auspices: Lay Board and Medical Advisory Committee
    Administrative Head: Kenneth O. Johnson, Ph.D., AH
    Medical Supervisor: Medical Advisory Committee

12. Stanford, Stanford University Speech and Hearing Clinic
    Auspices: Stanford University
    Administrative Head: Virgil A. Anderson, Ph.D., AS; Hayes A. Newby, Ph.D., AH
    Medical Supervisor: Community Advisory Board

13. Stockton, Speech and Hearing Center, College of the Pacific, Pacific Ave.
    Auspices: College of the Pacific
    Administrative Head: Howard L. Runion, Ph.D., AS
    Medical Supervisor: Group of Consultant Otolaryngologists

COLORADO

14. Denver, Children's Hospital Speech Clinic, Children's Hospital, E. 19th Ave., at Downing St.
    Auspices: Children's Hospital Association
    Administrative Head: Ruth M. Anderson, M.A., AS
    Medical Supervisor: Jean L. McMahon, M.D.

15. Denver, Denver Hearing Society, 1556 Emerson St.
    Auspices: Mile High United Fund
    Administrative Head: Elizabeth E. Kaho, Ph.D.
    Medical Supervisor: Doctors on Volunteer Board of Directors

16. Denver, University of Denver Hearing Center, University Park
    Auspices: Clinical Services, University of Denver
    Administrative Head: Mrs. Marion P. Downs, M.A., BH
    Medical Supervisor: Eleanor Botha, M.D.

17. Fort Collins, Speech and Hearing Center, Colorado State University
Auspices: Colorado State University
Administrative Head: William R. Leith, Ph.D., M
Medical Supervisor: None

CONNECTICUT

18. Hartford, Hartford Hearing League, Inc., 252 Asylum St.
Auspices: Greater Hartford Community Chest
Administrative Head: Mrs. Helen S. Lowry, Executive Director, B.A.
Medical Supervisor: Consulting Otologists

19. New Haven, Hearing and Speech Center, Yale-New Haven Medical Center, 789 Howard Ave.
Auspices: Yale University School of Medicine and Grace-New Haven Community Hospital
Administrative Head: Harris I. Pomerantz, M.S.
Medical Supervisor: John A. Kirchner, M.D.
New Haven †

DELAWARE

20. Wilmington, Delaware Hospital Audiology and Speech Center, 501 W. 14th St.
Auspices: Private General Hospital
Administrative Head: L. LeRoy Horne, M.A., BH
Medical Supervisor: Edgar N. Johnson, M.D.

DISTRICT OF COLUMBIA

21. Washington, D. C., Audiology and Speech Center, Walter Reed Army Hospital
Auspices: U. S. Army
Administrative Head: James P. Albrite, Major (MC), M.D.
Medical Supervisor: James P. Albrite, Major (MC), M.D.

22. Washington, D. C., Hearing and Speech Department, Children's Hospital of D. C., 2125 13th St. N. W.
Auspices: Children's Hospital
Administrative Head: Mrs. Edna K. Monsees, B.S., M
Medical Supervisor: Allan E. Walker Jr., M.D.

23. Washington, D. C., Leroy L. Sawyer Hearing and Speech Center, 1147 15th St., N. W.
Auspices: Episcopal Eye, Ear and Throat Hospital
Administrative Head: Lorraine Amos Roblee, M.A., BS, AH
Medical Supervisor: Frasier Williams, M.D.

*24. Washington, D. C., Hearing Rehabilitation Center, 1911 R St., N. W.
Auspices: Private
Administrative Head: Mrs. Edna K. Monsees, B.S., M
Medical Supervisor: Referring Otologists

25. Washington, D. C., Acoustical Research Audiology Unit, Munitions Building, 21st and Constitution Ave., N. W.
Auspices: Veterans Administration
Administrative Head: G. Donald Causey, Ph.D., BH
Medical Supervisor:

26. Washington, D. C., Washington Hearing Society, 1934 Calvert St., N. W.
Auspices: United Fund
Administrative Head: Ruth A. Hudnut, M.A.
Medical Supervisor: Consultant Otolaryngologists

† See also No. 153.

FLORIDA

27. Gainesville, Speech and Hearing Clinic, 321 Administration Building, University of Florida
Auspices: Florida Center of Clinical Services
Administrative Head: McKenzie W. Buck, Ph.D., AS
Medical Supervisor: Consultant Area Otologists

28. Pass-A-Grille (St. Petersburg), Audiology and Speech Correction Clinic, Veterans Administration Regional Office
Auspices: Veterans Administration
Administrative Head: R. Donald Bitler, M.A., M
Medical Supervisor: August Gehrke, M.D.

*29. St. Petersburg, Sertoma Speech Correction Clinic, American Legion Hospital for Crippled Children
Auspices: Sertoma Club
Administrative Head: R. Donald Bitler, M.A., M
Medical Supervisor: Members of Advisory Board

GEORGIA

30. Atlanta, Audiology and Speech Correction Clinic, Veterans Administration, 441-449 W. Peachtree St., N. E.
Auspices: Veterans Administration
Administrative Head: Allan J. Heffler, Chief, M.A., BH
Medical Supervisor: None

31. Atlanta, Davison School of Speech Correction, 1780 N. Decatur Rd., N. E.
Auspices: Private
Administrative Head: Louise D. Davison, B.A., AS, F
Medical Supervisor: Lester Brown, M.D.; Calhoun McDougal, M.D.

32. Atlanta, Junior League School for Speech Correction, Inc., 2020 Peachtree Rd., N. W.
Auspices: Junior League
Administrative Head: Claude S. Hayes, Ph.D., AH
Medical Supervisor: None

ILLINOIS

33. Carbondale, Speech and Hearing Clinic, Southern Illinois University
Auspices: Southern Illinois University
Administrative Head: I. P. Brackett, Ph.D., AS
Medical Supervisor: None

34. Champaign, University Hearing Center, University of Illinois, 321 Illini Hall
Auspices: University of Illinois
Administrative Head: E. Thayer Curry, Ph.D., AH
Medical Supervisor: Area Otologists

35. Chicago, Audiology and Speech Correction Clinic, Veterans Administration West Side Hospital, 820 South Damen Ave.
Auspices: Veterans Administration
Administrative Head: E. O. Windrem, M.A., BS
Medical Supervisor: J. B. Berardi, M.D.

36. Chicago, Chicago Hearing Society, 30 W. Washington St.
Auspices: Community Fund Agency
Administrative Head: Mary L. Thompson, Executive Director, M.A.
Medical Supervisor: Consulting Otologists

37. Chicago, Children's Memorial Speech and Hearing Clinic, 707 Fullerton Ave.
Auspices: Children's Memorial Hospital
Administrative Head: Miss Alice Saar, Director of Clinics, B.S.
Medical Supervisor: George S. Livingston, M.D.

38. Chicago, Hearing and Speech Service, Michael Reese Hospital, 29th and S. Ellis Ave.
Auspices: Michael Reese Hospital Medical Center
Administrative Head: Peter A. Campanelli, M.A.
Medical Supervisor: Robert Henner, M.D.

39. Chicago, Northwestern University Hearing Clinic, 303 E. Chicago Ave.
Auspices: Department of Otolaryngology, Medical School
Administrative Head: Raymond Carhart, Ph.D., F, AS, AH
Medical Supervisor: George E. Shambaugh Jr., M.D.; Eugene Derlacki, M.D.

40. Chicago, Speech and Hearing Center, Presbyterian Hospital, 1753 W. Congress St.
Auspices: Presbyterian Hospital
Administrative Head: Duncan R. C. Scott, M.A., BS, BH
Medical Supervisor: Stanton A. Friedberg, M.D.

41. Chicago, Illinois Speech and Hearing Center, 904 W. Adams St.
Auspices: Illinois Eye and Ear Infirmary, University of Illinois College of Medicine
Administrative Head: Charles Lightfoot, Ph.D., M
Medical Supervisor: Richard A. Buckingham, M.D.

42. Chicago, St. Luke's Speech and Hearing Service, 1439 S. Michigan Ave.
Auspices: St. Luke's Hospital
Administrative Head: William F. Waldrop, M.A., AH
Medical Supervisor: Paul H. Holinger, M.D.

43. Chicago, Speech Clinic and Audiology Unit, University of Chicago Clinics, 950 E. 59th St.
Auspices: University of Chicago Clinics
Administrative Head: Ralph F. Naunton, M.D.; Joseph Wepman, Ph.D., F, AS
Medical Supervisor: Ralph F. Naunton, M.D.

44. Evanston, Northwestern University Hearing Clinics, Adult, Speech Annex
Auspices: School of Speech, Northwestern University
Administrative Head: Raymond Carhart, Ph.D., AH, AS, F
Medical Supervisor: George E. Shambaugh Jr., M.D.; Eugene Derlacki, M.D.

45. Evanston, Institute for Language Disorders in Children, Speech Annex, Northwestern University
Auspices: Northwestern University
Administrative Head: Helmer R. Myklebust, Ed.D., F
Medical Supervisor:

46. Normal, Hearing Laboratory, Illinois State Normal University
Auspices: Illinois State Normal University
Administrative Head: Glenn Taylor, Ph.D., AH
Medical Supervisor: C. W. Hardy, M.D., and Local Otologists

47. Peoria, Bradley University Speech Clinic
Auspices: Bradley University
Administrative Head: Clara K. Mawhinney, Ph.D., AS
Medical Supervisor: Harold Vonachen, M.D., Morris Cohen, M.D.

INDIANA

48. Bloomington, Speech and Hearing Clinic, Indiana University
Auspices: Indiana University
Administrative Head: Robert Milisen, Ph.D., AS, F
Medical Supervisor: Bryan Quarles, M.D.

49. Indianapolis, Audiology and Speech Clinic, Indiana University Medical Center
Auspices: Indiana University Medical Center and Department of Otorhinolaryngology and Bronchoesophagology
Administrative Head: Francis L. Sonday, Director, Ph.D., AH
Medical Supervisor: Marlow W. Manion, M.D.; David E. Brown, M.D.

50. Lafayette, Purdue Speech and Hearing Clinic, Purdue University
Auspices: Purdue University
Administrative Head: Mack D. Steer, Ph.D., L.L.D., F, AS
Medical Supervisor: R. Calvert, M.D.; E. L. Van Buskirk, M.D.; L. W. Combs, M.D.

51. Muncie, Ball State Teachers College Out-Patient Clinic
Auspices: Ball State Teachers College
Administrative Head: Alan Huckleberry, Ph.D.
Medical Supervisor: W. G. Pippenger, M.D.

52. Terre Haute, Special Education Clinic, Indiana State Teachers College
Auspices: Indiana State Teachers College
Administrative Head: Rutherford B. Porter, Director, Ed.D.
Medical Supervisor: John M. Gnudrod, M.D.

*No information received for 1957 listing.
*No information received for 1957 listing.
*No information received for 1957 listing.
*No information received for 1957 listing.

IOWA

53. Cedar Falls, Speech and Hearing Clinic, Iowa State Teachers College
Auspices: Iowa State Teachers College
Administrative Head: David Williams, Ph.D., BS
Medical Supervisor: V. D. French, M.D.

54. Des Moines, Des Moines Hearing and Speech Center, 615 Locust St.
Auspices: United Community Services
Administrative Head: William K. Ickes, M.S., BH
Medical Supervisor: Medical Advisory Committee of Otologists

55. Grinnell, Grinnell Speech Clinic, Grinnell College
Auspices: Grinnell College
Administrative Head: Mrs. Velma B. Hiser, M.A., AS
Medical Supervisor: None

*56. Iowa City, University of Iowa Speech and Hearing Clinic, East Hall
Auspices: University of Iowa
Administrative Head: James F. Curtis, Ph.D., F, AH
Medical Supervisor: D. M. Lierle, M.D.

57. Iowa City, University of Iowa Otolaryngology and Maxillo Facial Surgery, University Hospital
Auspices: University Hospital
Administrative Head: D. M. Lierle, M.D., J. L. Shapley, Ph.D., Co-directors, BH
Medical Supervisor: D. M. Lierle, M.D.

KANSAS

58. Kansas City, University of Kansas Medical Center, L. B. Spake Hearing and Speech Department
Auspices: University of Kansas Medical Center
Administrative Head: June Miller, Ed.D., AH
Medical Supervisor:

59. Wichita, Institute of Logopedics, 2400 Jardine Dr.
Auspices: University of Wichita
Administrative Head: Martin F. Palmer, Sc.D., F, AS
Medical Supervisor: C. T. Hinshaw, M.D.

KENTUCKY

60. Lexington, Audiology Clinic, University of Kentucky
Auspices: Department of Psychology and Council of Jewish Women
Administrative Head: Frank Kodman Jr., Ph.D., BH
Medical Supervisor: Medical Advisory Committee

LOUISIANA

61. New Orleans, Tulane Speech and Hearing Center, 1430 Tulane Ave.
Auspices: Tulane University Medical School
Administrative Head: Jeannette K. Laguaite, Ph.D., AS
Medical Supervisor: J. W. McLaurin, M.D.

MAINE

62. Portland, Portland Hearing Society, Inc., 723 A Congress St.
Auspices: United Fund
Administrative Head: Miss Pauline G. Carter, B.A.
Medical Supervisor: Advisory Board of Consulting Otologists

MARYLAND

63. Baltimore, The Baltimore Hearing Society, Inc., 928 N. Charles St.
Auspices: Community Chest of Baltimore
Administrative Head: Jay Cherry, M.A., BS
Medical Supervisor: None

64. Baltimore, The Hearing and Speech Center, The Johns Hopkins Medical School—Hospital
Auspices: The Johns Hopkins Medical Institutions
Administrative Head: William G. Hardy, Ph.D., AH, F
Medical Supervisor: John E. Bordley, M.D.

65. Baltimore, University of Maryland, Department of Otolaryngology, University Hospital
Auspices: University of Maryland
Administrative Head: Cyrus L. Blanchard, M.D.
Medical Supervisor: Cyrus L. Blanchard, M.D.

MASSACHUSETTS

66. Boston, Boston Guild for the Hard of Hearing, 283 Commonwealth Ave.
Auspices: Voluntary Health Agency; partial support of United Fund
Administrative Head: Mrs. Claire K. Kennedy
Medical Supervisor: New England Otolaryngological Association

67. Boston, Boston University Speech and Hearing Center, 332 Bay State Rd.
Auspices: Boston University
Administrative Head: Wilbert L. Pronovost, Ph.D., AS, F
Medical Supervisor: M. Stuart Strong, M.D.

68. Boston, Veterans Administration Audiology Clinic, 17 Court St.
Auspices: Veterans Administration
Administrative Head: George E. Hill Jr., M.A.
Medical Supervisor: Benjamin Bell, M.D.

69. Boston, Emerson College, Samuel D. Robbins Speech and Hearing Clinic, 145 Beacon St.
Auspices: Emerson College
Administrative Head: Catherine C. Perry, M.A., AS
Medical Supervisor: College Physician, and Medical and Educational Advisory Board

70. Boston, Hearing and Speech Clinic, Children's Medical Center, 300 Longwood Ave.
Auspices: Children's Medical Center
Administrative Head: Adam J. Sortini, Ed.D., BS, BH
Medical Supervisor: Board of three staff members

71. Boston, The Frederic Winthrop Foundation, Massachusetts Eye and Ear Infirmary, 243 Charles St.
Auspices: Massachusetts Eye and Ear Infirmary
Administrative Head: Alan S. Feldman, Ph.D., BS
Medical Supervisor: Philip E. Meltzer, M.D.

72. Springfield, Springfield Hearing League, Inc., 1694 Main St.
Auspices: United Fund, Inc., of Springfield
Administrative Head: Mrs. Grace F. Gaffney, A.B.
Medical Supervisor: Eugene Boss, M.D. Medical Advisory Board

73. Worcester, Worcester Hearing League, Inc., 306 Main St.
Auspices: Worcester Community Chest
Administrative Head: Miss Kathleen F. Kirby, M.A.
Medical Supervisor: Committee of Consulting Otologists

MICHIGAN

74. Ann Arbor, Speech Clinic, 1007 E. Huron St., University of Michigan
Auspices: University of Michigan
Administrative Head: H. Harlan Bloomer, Ph.D., AS, F
Medical Supervisor: None

75. Detroit, Henry Ford Hospital Hearing and Speech Clinic, 2799 W. Grand Blvd.
Auspices: Henry Ford Hospital
Administrative Head: A. Bruce Graham, Ph.D., BH
Medical Supervisor: J. Lewis Dill, M.D.

76. Detroit, Hearing Clinic, 656 W. Warren Ave.
Auspices: Wayne State University
Administrative Head: John H. Gaeth, Ph.D., AH
Medical Supervisor: Heinrich Kobrak, M.D., Ph.D.

77. East Lansing, Speech and Hearing Clinic, Michigan State University
Auspices: Michigan State University
Administrative Head: Ralph Leutenegger, Ph.D., BS
Medical Supervisor: Area Otologists as Consultants

78. Flint, Michigan School for the Deaf Speech and Hearing Clinic, Court St.
Auspices: State Board of Education
Administrative Head: Thomas H. Poulos, M.A., BH
Medical Supervisor: None

79. Grand Rapids, Grand Rapids Society for the Hard of Hearing, 1230 Madison Ave., S. E.
Auspices: United Community Chest of Grand Rapids and Kent County
Administrative Head: Isabelle Starkey, Director, M.A.
Medical Supervisor: Five M.D.s on Board of Directors

80. Kalamazoo, Constance Brown Society for Better Hearing, 316 Commerce Building
Auspices: Kalamazoo Foundation, Kalamazoo Community Chest
Administrative Head: Alfred R. Thea, M.S., AH
Medical Supervisor: Ralph B. Fast, M.D.

81. Lansing, Hearing Conservation Section, Michigan Department of Health, Old DeWitt Rd.
Auspices: Maternal and Child Health Division, Michigan Department of Health
Administrative Head: Courtney D. Osborn, Chief, Hearing Conservation Section, M.A., AH
Medical Supervisor: Goldie B. Corneliuson, M.D.

82. Lansing, Michigan Association for Better Hearing, 408 Hollister Building
Auspices: Michigan United Fund
Administrative Head: Stahl Butler, Executive Director, M.A.
Medical Supervisor: Advisory Board

83. Mt. Pleasant, Central Michigan College
Auspices: Central Michigan College
Administrative Head: Wilbur E. Moore, Dean, Psycho-Educational Services, Ph.D., AS, F
Medical Supervisor: None

MINNESOTA

*84. Minneapolis, Audiology Clinic, University of Minnesota Hospitals
Auspices: University of Minnesota
Administrative Head: Frank M. Lassman, Ph.D., AH
Medical Supervisor: Lawrence R. Boies, M.D.

85. Minneapolis, University of Minnesota Speech and Hearing Clinic
Auspices: University of Minnesota
Administrative Head: Ernest H. Henrikson, Ph.D., F, AS
Medical Supervisor: Donald Cowan, M.D.

86. Rochester, Department of Audiology, Section of Otolaryngology and Rhinology, Mayo Clinic, 200 Second St., S. W.
Auspices: Mayo Clinic
Administrative Head: LeRoy D. Hedgecock, Ph.D., AS, AH
Medical Supervisor: Henry L. Williams, M.D.

MISSOURI

87. Columbia, University of Missouri Speech and Hearing Clinic, 325 Switzler Hall
Auspices: University of Missouri
Administrative Head: Charlotte G. Wells, Ph.D., AS
Medical Supervisor: Consultants at University Medical Center

88. Kansas City, Veterans Administration Regional Office, 1828 Walnut
Auspices: Veterans Administration
Administrative Head: Morton Zarcoff, M.S.
Medical Supervisor: A. L. Kelly, M.D.

89. St. Louis, Central Institute for the Deaf, 818 S. Kingshighway
Auspices: Central Institute for the Deaf
Administrative Head: S. Richard Silverman, Ph.D., AH, F
Medical Supervisor: Theo E. Walsh, M.D.; Harold Cutler, M.D.

NEBRASKA

90. Lincoln, Speech and Hearing Laboratories, University of Nebraska, Temple Building, 12th and R Sts.
Auspices: University of Nebraska
Administrative Head: John Wiley, Ph.D., AS
Medical Supervisor: Student Health Staff and Consultants from Veterans Administration

NEW JERSEY

91. Newark, Henry C. Barkhorn Memorial Hearing and Speech Center, 77 Central Ave.
Auspices: Newark Eye and Ear Infirmary
Administrative Head: Herbert E. Rickenberg, M.A., AS, BH
Medical Supervisor: William H. Hahn, M.D.

92. Newark, St. Barnabas Hospital Rehabilitation Center, 701 N. High St.
Auspices: St. Barnabas Hospital
Administrative Head: Lyndon Peer, M.D.
Medical Supervisor: Franklin Keim, M.D.

NEW MEXICO

93. Sante Fe, Hearing Test and Child Study Center, 1060 Cerrillos Rd.
Auspices: New Mexico School for the Deaf
Administrative Head: Jack A. Willeford, B.A., BS, BH
Medical Supervisor: James McCrory, M.D.

NEW YORK

94. Buffalo, Buffalo Hearing and Speech Center, Inc., 2183 Main St.
Auspices: Buffalo Otolaryngological Society and University of Buffalo
Administrative Head: Elmo L. Knight, M.S., BH
Medical Supervisor: Herrmann E. Bozer, M.D.

95. Buffalo, Children's Hospital Hearing and Speech Center, 219 Bryant St.
Auspices: New York State Association for Crippled Children
Administrative Head: Elmo L. Knight, M.S., BH
Medical Supervisor: Herrmann E. Bozer, M.D.

96. Mineola, Long Island Hearing and Speech Society, Inc., First St., Mineola, Long Island, New York
Auspices: Nonprofit organization
Administrative Head: Mrs. Mary G. Fuller
Medical Supervisor: Kenneth B. Brown, M.D.

97. New York, Audiology Clinic, Veterans Administration, 252 Seventh Ave.
Auspices: Veterans Administration
Administrative Head: Martin Cohen, M.A., BS, Chief Audiologist
Medical Supervisor: Morris F. Heller, M.D.

98. New York, Speech and Hearing Clinic, Columbia Presbyterian Medical Center, 622 W. 168th St.
Auspices: The Presbyterian Hospital of New York City
Administrative Head: Maurice H. Miller, Ph.D., AS, BH
Medical Supervisor: Edmund P. Fowler Jr., M.D.

99. New York, Hearing and Speech Center, Bellevue Hospital, 1st Ave. and 27th St.
Auspices: New York University—Bellevue Medical Center
Administrative Head: Donald M. Markle, M.Ed., AH
Medical Supervisor: John F. Daly, M.D.

*100. New York, Institute of Physical Medicine and Rehabilitation, Speech and Hearing Therapy Department, 400 E. 34th St.
Auspices: New York University Bellevue Medical Center
Administrative Head: Martha L. Taylor, M.A., AS
Medical Supervisor: John Daly, M.D.

101. New York, Hearing and Speech Center, Manhattan Eye, Ear and Throat Hospital, 210 E. 64th St.
Auspices: Manhattan Eye, Ear and Throat Hospital
Administrative Head: Mary Wood Whitehurst, M.A., M
Medical Supervisor:

*102. New York, Department of Audiology and Hearing Research Center, New York Eye and Ear Infirmary, 218 Second Ave.
Auspices: New York Eye and Ear Infirmary
Administrative Head: J. A. DePew Jr.
Medical Supervisor: J. Swift Hanley, M.D.

103. New York, Hunter College Speech and Hearing Center, 695 Park Ave.
Auspices: Hunter College of the City of New York
Administrative Head: Moe Bergman, Ed.D., AH
Medical Supervisor: None

104. New York, Speech and Hearing Center, 525 E. 68th St.
Auspices: The New York Hospital—Cornell Medical Center
Administrative Head: Miss Margaret Soisson, M. A., M
Medical Supervisor: James A. Moore, M.D.

105. New York League for the Hard of Hearing, 480 Lexington Ave.
Auspices:
Administrative Head: Eleanor C. Ronnei, M.A.
Medical Supervisor:

106. New York, Queens Speech and Hearing Center, Queens College, Flushing 67, N. Y.
Auspices: Queens College
Administrative Head: Jon Eisenson, Ph.D., AS
Medical Supervisor: Charles Seelig, M.D.

107. New York, Hearing and Speech Department, Amsterdam Ave. and 113th St.
Auspices: St. Luke's Hospital
Administrative Head: Erik Stenersen
Medical Supervisor: James C. Kirkbright, M.D., F.A.C.S.

108. Rochester, Hearing and Speech Center of Rochester, Inc., 501 Main St., W.
Auspices: Rochester Community Chest
Administrative Head: Irwin Brown, Ph.D., AS
Medical Supervisor: George M. Trainor, M.D.

109. Syracuse, Gordon D. Hoople Hearing and Speech Center, Syracuse University 805 S. Crouse Ave.
Auspices: Syracuse University
Administrative Head: Louis M. DiCarlo, Ed.D., F, AS, AH
Medical Supervisor: Gordon D. Hoople, M.D.

110. Utica, Utica Hearing and Speech Center, Children's Hospital Home
Auspices: Children's Home and American Association for Crippled Children
Administrative Head: Karl Gruppe, M.D.
Medical Supervisor: Karl Gruppe, M.D.

NORTH CAROLINA

111. Winston-Salem, Hearing and Speech Center, N. C. Baptist Hospitals, Inc., South Hawthorne Rd.
Auspices: Department of Otolaryngology, Bowman Gray School of Medicine
Administrative Head: Malcolm B. McCoy, Ph.D., BH
Medical Supervisor: James A. Harrill, M.D.

OHIO

112. Athens, Ohio University Speech and Hearing Clinic
Auspices: Ohio University
Administrative Head: A. C. LaFollette, Ph.D., AS
Medical Supervisor: Eleanora L. Schmidt, M.D.

113. Bowling Green, Speech and Hearing Clinic, Bowling Green State University
Auspices: Bowling Green State University
Administrative Head: Melvin Hyman, Ph.D., AS
Medical Supervisor: None

114. Cincinnati, Cincinnati Speech and Hearing Center, 3006 Vernon Pl.
Auspices: Community Chest and Council of the Cincinnati Area
Administrative Head: Warren E. Johnson, Director, M.A., BH
Medical Supervisor: Samuel Seltz, M.D.; Raymond Hilsinger, M.D.

115. Cleveland, The Cleveland Hearing and Speech Center, 11206 Euclid Ave.
Auspices: Cleveland Welfare Federation and Western Reserve University
Administrative Head: George J. Fortune, Director, M.A.
Medical Supervisor: Medical Committee

116. Columbus, Columbus Hearing Society, 209 S. High St.
Auspices: Private
Administrative Head: Samuel M. Cohen, M.Ed., BS
Medical Supervisor: Otological Advisory Board

117. Columbus, Ohio Department of Health Hearing Conservation Unit, 306 Ohio Departments Building
Auspices: Child Hygiene Division, Ohio Department of Health
Administrative Head: William A. Grimm, Chief, Hearing Conservation Unit, M.A.
Medical Supervisor: Margot D. Hartmann, M.D.

118. Columbus, Ohio State University Speech and Hearing Clinic, Room 300, Derby Hall
Auspices: Ohio State University
Administrative Head: Henry M. Moser, Ph.D., AS
Medical Supervisor: William H. Saunders, M.D.

119. Kent, Kent State University Speech and Hearing Clinic
Auspices: Kent State University
Administrative Head: John R. Montgomery, Director, M.A., AS
Medical Supervisor: Richard Stahl, M.D., Medical Consultant

120. Toledo, Toledo Hearing League, 2313 Ashland Ave.
Auspices: Private—Community Chest Agency
Administrative Head: Mrs. George Reisbach
Medical Supervisor: Advisory Board of Otologists

121. Youngstown, Youngstown Hearing and Speech Center, 69 Illinois Ave.
Auspices: Voluntary Agency, Community Chest Support; American Hearing Society
Administrative Head: May Vetterle, M.A., BH
Medical Supervisor: Medical Committee

OKLAHOMA

122. Oklahoma City, Oklahoma University Speech and Hearing Clinic, 824 E. 14th St.
Auspices: University of Oklahoma
Administrative Head: John W. Keys, Ph.D., AH
Medical Supervisor: O. A. Watson, M.D., and Chester McHenry, M.D.

OREGON

123. Portland, Portland Center for Hearing and Speech, 2111 N. E. Weidler
Auspices: Private Agency Affiliated with United Fund
Administrative Head: George J. Leshin, M.Ed., BH
Medical Supervisor: David D. DeWeese, M.D.

PENNSYLVANIA

124. Harrisburg, Commonwealth Speech and Hearing Rehabilitation Center
Auspices: State Department of Public Instruction
Administrative Head: Gladys B. Fish, M.Ed., BS
Medical Supervisor: None

125. Lancaster, Hearing Conservation Center of Lancaster County, Pa., 427 N. Duke St.
Auspices:
Administrative Head: Juliet G. Stephan, R.N.
Medical Supervisor: Lloyd S. Hutchison, M.D.

126. Philadelphia, Hospital of the University of Pennsylvania Speech and Hearing Center
Auspices: Hospital of the University of Pennsylvania
Administrative Head: Martin C. Schultz, Ph.D., BH, BS
Medical Supervisor: Harry P. Schenck, M.D.

127. Philadelphia, Commonwealth Speech and Hearing Rehabilitation Center, St. Christopher's Hospital for Children
Auspices: State Department of Public Instruction
Administrative Head: Milton N. Brutten, Ph.D.
Medical Supervisor: None

128. Philadelphia, Audiology Section, Temple University Medical Center, 3401 N. Broad St.
Auspices: Department of Otorhinology
Administrative Head: Philip E. Rosenberg, Ph.D., BH
Medical Supervisor: David Myers, M.D., and Staff

129. Philadelphia, National Naval Aural Rehabilitation Center, 17th and Pattison Ave.
Auspices: U. S. Navy
Administrative Head: Joseph A. Scanlon, Coordinator of Services, A.B.
Medical Supervisor: Capt. William C. Livingood, (MC) USN

130. Pittsburgh, Pittsburgh Hearing Society, 313 Sixth Ave.
Auspices: Voluntary Health Agency, partially supported by United Fund
Administrative Head: Miss Phyllis McCalmont, M.A., M
Medical Supervisor: Professional Advisory Committee

131. Pittsburgh, Commonwealth Speech and Hearing Rehabilitation Center, Eye and Ear Hospital, 230 Lothrop St.
Auspices: Department of Public Instruction
Administrative Head: Elmer Owens, Audiologist, M.A., M
Medical Supervisor: None

132. Pittsburgh, Department of Audiology, Eye and Ear Hospital, 230 Lothrop
Auspices: University of Pittsburgh
Administrative Head: Leo G. Doerfler, Ph.D., F, AH
Medical Supervisor: Kenneth Day, M.D.

133. Reading, Hearing Clinic, Reading Hospital
Auspices: Reading Hospital
Administrative Head: James E. Landis, M.D.
Medical Supervisor:

134. Scranton, Commonwealth Speech and Hearing Rehabilitation Center, 1800 Washington
Auspices: Department of Public Instruction
Administrative Head: Albert F. Johnson, Audiologist, M.A., BH
Medical Supervisor: None

135. University Park, Commonwealth Speech and Hearing Rehabilitation Center, Pennsylvania State University
Auspices: Department of Public Instruction
Administrative Head: Raymond J. Lezak, M.A., BS, BH
Medical Supervisor: James M. Campbell Jr., M.D.

136. University Park, Speech and Hearing Clinic, Pennsylvania State University
Auspices: Pennsylvania State University
Administrative Head: E. T. McDonald, Ed.D., AS
Medical Supervisor: Tom Mebane, M.D.

SOUTH CAROLINA

137. Greenville, Greenville Hearing and Speech Center, General Hospital
Auspices: United Fund of Greenville County
Administrative Head: Mervyn L. Falk, M.A., BS
Medical Supervisor: A. J. Goforth, M.D.

TENNESSEE-VIRGINIA

138. Bristol, Bristol Speech and Hearing Center, Bristol Memorial Hospital
Auspices: The Border Guild and Department of Public Health of Tennessee
Administrative Head: Clarence E. Webb, Ph.D., BS
Medical Supervisor: Fred McCall, M.D.

TENNESSEE

139. Chattanooga, Speech and Hearing Center of Chattanooga-Hamilton County, 605 Vine St.
Auspices: Nonprofit
Administrative Head: J. Dale Welsch, Ed.D., AS
Medical Supervisor: Charles Harold Alper, M.D.

140. Memphis, Memphis Speech and Hearing Center, Inc., 874 Monroe Ave.
Auspices: University of Tennessee College of Medicine
Administrative Head: John A. Irwin, Director, M. A.
Medical Supervisor: Sam Sanders, M.D.

141. Nashville, The Bill Wilkerson Hearing and Speech Center, 19th Ave. South at Edgehill, or Vanderbilt University Hospital
Auspices: Nonprofit Corporation
Administrative Head: Freeman McConnell, Ph.D., AH
Medical Supervisor: W. W. Wilkerson Jr., M.D.

TEXAS

142. Austin, University of Texas Speech and Hearing Clinic, Department of Speech, University of Texas
Auspices: The University of Texas
Administrative Head: Jesse J. Villarreal, Ph.D., AS
Medical Supervisor: Douglas Barkley, M.D.

143. Dallas, Audiology and Speech Correction Clinic, Veterans Administration Hospital, 4500 S. Lancaster Rd.
Auspices: Veterans Administration
Administrative Head: Vernon C. Bragg, Ph.D.
Medical Supervisor: Ludwig A. Michael, M. D.

144. Galveston, Hearing and Speech Clinic, The University of Texas Medical Branch, 105 Kieller Building
Auspices: The University of Texas Medical Branch
Administrative Head: C. Olaf Haug, M.A., AH
Medical Supervisor: Fred R. Guilford, M.D.

145. Houston, Houston Speech and Hearing Center, 6504 Bertner Ave.
Auspices: United Fund of Houston, Harris County
Administrative Head: Jack L. Bangs, Ph.D., AS, AH
Medical Supervisor: Committee from Local County Society

UTAH

146. Salt Lake City, Speech and Hearing Center, University of Utah, 1699 E. Fifth S. (Hempstead Rd.)
Auspices: University of Utah
Administrative Head: Wallace A. Goates, Ph.D., AH, AS
Medical Supervisor: David A. Dolowitz, M.D.

VERMONT

*147. Rutland, Children's Rehabilitation Center, 88 Park St.
Auspices: Vermont Association for the Crippled, Inc.
Administrative Head: Miss Harriet M. Dunn, M.A., AS, Director of Speech and Hearing Services
Medical Supervisor: Ralph H. Seeley Jr., M.D.

VIRGINIA

148. Charlottesville, Speech and Hearing Center, University of Virginia, 109 New Cabell Hall
Auspices: University of Virginia
Administrative Head: James M. Mullendore, Ph.D., AS
Medical Supervisor: Fletcher D. Woodward, M.D.

149. Richmond, Hearing and Speech Center, Medical College of Virginia, 12th and Broad Streets
Auspices: Medical College of Virginia
Administrative Head: S. James Cutler (Hearing), B.S., M; John Collison, (Speech), M.A., AS
Medical Supervisor: P. N. Pastore, M.D.

WASHINGTON

150. Seattle, Seattle Hearing and Speech Center, 1229 10th Ave. N.
Auspices:
Administrative Head: Clyde E. Mott, Executive Director, B.A.
Medical Supervisor: John Tolan, M.D.

151. Spokane, Inland Speech and Hearing Clinic, 304 Paulsen Building
Auspices:
Administrative Head: John W. Veatch, B.A., BS
Medical Supervisor: None

WISCONSIN

152. Milwaukee, Hearing Evaluation Center, 3203 N. Downer Ave.
Auspices: University of Wisconsin
Administrative Head: Robert J. Briskey, M.A., BS
Medical Supervisor: Meyer S. Fox, M.D.

Addendum

153. Connecticut, New Haven, The Audiology Center, 33 Trumbull St.
Auspices: Private
Administrative Head: Norton Canfield, M.D., AH, F
Medical Supervisor: Norton Canfield, M.D.

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Brief-Tone Audiometry

Temporal Integration in the Hypacusic

J. DONALD HARRIS, Ph.D.; HENRY L. HAINES, M.D., and CECIL K. MYERS, B.S., New London, Conn.

Introduction

Authors whose work is too extensive to summarize here have dealt with the problem of how the threshold of hearing changes when the stimulus tone is made shorter and shorter. Much is also known about the effect of tone duration upon loudness (of which the threshold is a limiting case). We now have access to copious, though not complete, data on loudness and on threshold as affected by frequency, band width, intensity, duration, and repetition rate, much of it from the recent papers of Garner.[2,3]

Perhaps the major point of theoretical interest in this general area is how and under what conditions the ear integrates energy. In other terms, whether, for any given set of conditions, all the energy present in a stimulus over any period of time is utilized by the ear, or whether some part of that energy is wasted. Energy is wasted if it does not contribute to a loudness increase or to an improvement in threshold. A very practical result might be expected to follow in differential diagnosis, if it could be demonstrated that certain types of deafness exhibit integration characteristics different from the normal case.

A first indication that an ear with intracochlear disturbance might yield atypical integration data may be inferred from a study by de Maré.[8] He showed in three patients with either syphilis, presbycusis, or progressive perceptive deafness of unknown origin that the spread of adaptation, or "residual masking," was much greater, up and down the basilar membrane, than was the case with normal ears. Lüscher and Zwislocki[7] at about the same time confirmed and extended this observation in a patient with acoustic trauma. While it should be cautioned[4] that the sensation level used for the deafened ear cannot be directly compared with the normal case, nevertheless a real effect is probably present. Thus, Lüscher and Zwislocki give their patient's binaural loudness-matching data at 6700 cps, from which it can be seen that a sensation level of 40 db. on the deafened side is loudness matched with 65 db. on the normal side. Now with a stimulating tone of 6700 cps at 65 db. sensation level in the normal ear, a threshold shift of about 7 db. remains at 10 kc.; while in the deafened ear, with the same tone at 40 db. (i. e., equal loudness) the threshold shift at 10 kc. also amounts to about 7 db.

In other words, for equal loudness the spread of frequency effect is similar for the defective and the normal ear, but certainly not so for equal level above threshold. What we have here is an indication that for at least some deafnesses, a certain decibel level above threshold brings into play a fiber area considerably wider than normal on the basilar membrane. Now note that this "residual fatigue" effect has been tied to processes within the cochlea, and further that we have shown this effect and "recruitment" to be connected with one and the same intracochlear tissue.[5] With these three pieces of information, we can begin to see that, if a tone at a certain intensity actuates more fibers than normal, these fibers could add their bit to the perceived loudness, and recruitment could arise. And, in addition, one might guess that in such a condition there would be an appreciable effect on energy

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integration (which is simply some sort of storage over a duration of time).

Miskolczy-Fodor [9,10] has recently documented just this assumption, that recruitment (actually an improvement in the way the ear handles intensities) coexists pari passu with an improvement in the way the ear handles durations. Certain summary figures and nomographs of Miskolczy-Fodor deserve the widest circulation and discussion.

Perdoncini [11] also has some preliminary observations on the effect of audiometry with brief tones. His technique is to obtain an audiogram with the usual duration of signal, and then another audiogram with a signal of 15 msec. duration. Discrepancies between the two curves seem to bear upon the results of speech audiometry.

Certain of Miskolczy-Fodor's results in the clinic have been reproduced in the laboratory by Jerger, using ears temporarily fatigued by loud tones.[6]

Eisenberg [1] has studied the Miskolczy-Fodor effect very extensively in the clinic and reports it to be useful. Audiologists would profit from a prompt publication of Miss Eisenberg's excellent study.

The present paper studies energy integration under conditions similar to those used by Miskolczy-Fodor and several earlier workers, and particularly we consider the use of disturbances of integration as a clinical test, both according to the method and index proposed by Miskolczy-Fodor and according to other indices which we have found convenient.

Apparatus and Procedure

(*a*) *Selection of Patients.*—Since we wished to add further data on the relation between energy integration and recruitment, we studied a group of normal ears and then with special care we studied numerous patients on most of whom we had available at least one normal or near-normal frequency to serve as a reference for loudness balancing, and also to serve as a norm for integration data. We regard as extremely important the principle of the patient serving as his own control in exploratory work of this sort. Reference to normal ears is often insufficient.

(*b*) *Threshold vs. Duration of Stimulus.*—The output of a high-quality oscillator was set to a desired frequency and led through a shaper amplifier (with associated timer), a bank of 1-db.-step attenuators, and a narrow-bandpass filter to the patient's earphone, a monaural Permoflux PDR-8 unit. The timer was set to deliver a sequence of tones of any duration from 5 through 500 msec., alternating with a 0.6 sec. interval of silence. In an effort to keep the frequency content of the tonebursts confined so far as possible to the stated oscillator frequency, the shaper amplifier was adjusted to give a 5 msec. rise-fall characteristic to the onset and termination of the single burst.

The patient was asked to listen to a series of bursts, and to turn an attenuator until he could hear about half the bursts. After a few practice trials, when he felt confident he knew what do do and what to listen for, and when his practice trials indicated reasonable consistency, the patient was required to make four to six adjustments to threshold. All the attenuator settings were noted and averaged for that duration. A new duration was then selected, and so on. Thus a graph could be drawn for every patient for every frequency used, relating absolute threshold to tonal duration. This graph was used to denote the "slope of integration" at each frequency used for each patient. The "Slope of Integration" is the number of decibels threshold shift occurring as the duration of the stimulus is changed by 1 log unit (for example, from 150 msec. to 15 msec.)

(*c*) *Loudness-Balancing.*—We used either binaural or monaural matching, as the case allowed. Data were entered on graph paper in the common fashion, which yields a 45 degree diagonal for no recruitment; each curve was assessed for which type of recruitment was exhibited, whether quick and complete, asymptotic or straight-line, slight, delayed, or any combination.

(*d*) *Background Data.*—Complete ac-bc audiograms were taken with continually calibrated equipment, and with proper care as to techniques of masking. The Sonotone bc vibrator was spring-held against the mastoid with a special helmet, and had been calibrated on a dozen normal ears. An audiological history and otologist's notes were also at hand.

Results

Complete otoaudiological notes, together with complete audiometry and data on tests of recruitment and of energy integration are on file. We sought to know whether energy integration could throw light on the locus of disorder in a defective ear, and so to be useful as an adjunct in diagnosis. To this end, integration results have been assessed for every patient, from the point of view whether such data are worth the cost in time and effort. In the following sharply abbreviated case histories, we present only such information as bears upon this matter.

Patients 1 and 2 substantiate the fact that pure conductive peripheral deafness never exhibits abnormalities in integration data. The next four (Patients 3, 4, 5, 6) show that noise trauma is always accompanied by disturbance of integration, roughly in proportion to audiometric loss. The next three (7, 8, 9) show how integration data can help pick up a nonperipheral defect overlaid on a preponderantly peripheral conductive loss.

The next eight patients (10, 11, 12, 13, 14, 15, 16, 17) represent a most important group in which it is shown that lesions within the cochlea may or may not yield atypical integration data, and therefore that such data may significantly bear upon the locus of lesion and even upon the diagnosis. Patient 10 shows how normal integration data can exist in intracochlear deafness, and thus help to rule out acoustic trauma. Patient 11 shows that nontraumatic organ of Corti lesions can distort integration (though not accompanied by perstimulatory adaptation). Patient 12 shows that integration data can help exclude peripheral conductive deafness from a lesion undetectable by otoscopy, and help to assign the locus of lesion to the cochlea but not the organ of Corti. Patient 13 shows that some intracochlear disorders in addition to acoustic trauma can yield atypical integration data. Patient 14 shows that some types of cochlear defect, even those exhibiting recruitment, nevertheless can yield normal integration data. Patient 15 shows that certain as yet unspecified cochlear lesions (not of the organ of Corti) can furnish normal integration data, while in the same cochlea an additional defect of another type can produce atypical integration data at other frequencies. Patient 16 shows that somewhat atypical integration data can be produced by supposedly cochlear disorders not of the organ of Corti. Patient 17 shows how integration data can be used to help rule out central nervous system lesion and to help place the defect within the cochlea, but not in the organ of Corti.

The significance of the above group of eight patients, and others like them, is that a hope is offered that by studying integration data in connection with data from other tests, such as bc, recruitment, perstimulatory adaptation, and others, it may be possible to determine the particular test profiles yielded by particular intracochlear disorders. A start has been made on presbycusis, acoustic trauma, and Ménière's, and data are beginning to come in on other disorders.

The next two patients (18, 19) show that the absence of recruitment and atypical integration data are commonly exhibited together in presbycusis, and help to differentiate that disorder from certain other cochlear defects.

The next three patients (20, 21, 22), with preponderantly peripheral conductive deafness, show how atypical integration data can help to point out that a cochlear loss is beginning to overlay the conductive loss, and at what frequency region this begins.

The last three patients (23, 24, 25) are a miscellaneous group, showing, respectively, that a temporary conductive loss can yield

normal integration data which (after the patient's recovery) are found to be sharply abnormal as a result of a permanent cochlear deafness; that at lower frequencies an integration test may be more sensitive to cochlear deafness than is perstimulatory deafness, and finally that a combination of normal integration data with "garbled" speech may lead to a search for a central lesion.

PATIENT 1.—Age 22. Otoscopy entirely negative. No family history. Was told in school in Fifth Grade he had some hearing loss. No perstimulatory adaptation. Slight straight-line recruitment.

	250	500	1 kc.	2 kc.	4 kc.	8 kc.	
L ac	25	25	25	10	10	0	(R ear entirely normal)
bc	0	7.5	10	0			

Conclusion thus far: Long-duration stabilized peripheral conductive loss, cause unknown.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0 *	
R/500	11 db.	140 msec.	(Control)
R/1 kc.	8	200	(Control)
L/500	8	200	
L/1 kc.	7.5	200	

All data essentially normal.

*T_0=the duration, in milliseconds, above which no further improvement in threshold occurs.

This patient included to show confirmatory nature of integration data in pure peripheral conductive cases.

PATIENT 2.—Age 42. Otoscopy negative. Very slight progressive deafness. No recruitment. Slight if any perstimulatory adaptation.

	250	500	1 kc.	2 kc.	4 kc.	8 kc.	
R ac	40	35	30	20	35	25	
bc	0	2.5	0	15	5	—10	("Carhart notch"?)

Conclusion thus far: Uncomplicated monaural otosclerosis.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
L/1 kc.	10 db.	300 msec.	(Control)
R/1 kc.	10	150	

Integration data essentially normal.

This patient included to show reliability of observations, and consistency of interpretation in uncomplicated otosclerosis.

PATIENT 3.—Age 29. High-tone dip of long standing, probably traumatic from Air Force service. Otoscopy negative, bc and recruitment tests agree.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.	
L ac	—10	—10	—10	—5	25	45	15	15	(R ear similar)
bc	—5	—10	0	0	15	25	10	0	

Conclusion thus far: Probably traumatic lesion of organ of Corti.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
L/1 kc.	7.2 db.	150 msec.	(Control)
L/2 kc.	6.5	130	
L/3 kc.	6	160	
L/4 kc.	4.2	160	

Frequencies normally heard yield normal integration data; frequencies with audiometric loss yield somewhat flatter slopes in proportion to loss.

This patient included to confirm (for traumatic lesions at least) Miskolczy-Fodor's relation between slope of integration and extent of lesion on the organ of Corti.

PATIENT 4.—Age 31. History negative except for 1000 rounds of small arms fire and 13 years as a Diesel engineman. Otoscopy negative; bc and recruitment tests agree.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.
R ac	0	0	0	10	55	55	20	10
bc	5	10	10	10	55	60	..	..
L ac	0	0	0	15	55	65	65	35	10
bc	5	5	5	15	..	..	..	..	..

Conclusion thus far: Traumatic lesion of the organ of Corti.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/2 kc.	7 db.	160 msec.	(Control)
L/2 kc.	3	200	

Defective frequency yields extremely flat slope.

This patient included to show that traumatic end-organ lesion always yields atypical integration data.

PATIENT 5.—Age 45. Otoscopy negative. Former mining machine and trip-hammer operator. Quick and complete recruitment.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.	
R ac	—5	—5	—5	15	40	30	40	25	(L ear similar)
bc	0	0	2.5	10.5	..	28	..	..	

Conclusion thus far: Traumatic lesion of organ of Corti.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/1 kc.	12 db.	130 msec.	(Control)
R/2 kc.	3	400	
R/3 kc.	3.7	200	
R/4 kc.	5.7	200	

This patient included to confirm the existence of integration distortion in traumatic end-organ lesions.

PATIENT 6.—Age 25. Otoscopy negative. Diesel noise exposure for several years. No perstimulatory adaptation whatever. Recruitment quick and complete.

	250	500	1 kc.	2 kc.	4 kc.	8 kc.	
L ac	—5	0	20	10	35	0	(R ear similar)
bc			20				

Conclusion thus far: Typical end-organ loss of Dieselman.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
L/250	15 db.	200 msec.	(Control)
L/1 kc.	6.5	200	

Flatter integration slope at L/1 kc. agrees with bc and recruitment data.

This patient included to show that integration data and perstimulatory adaptation by no means always coincide.

PATIENT 7.—Age 28. Mastoidectomy three years previously. Chronic suppurative otitis occasionally, last episode in recent weeks. L drum scarred. A 1 kc. tone at 130 db. SPL is not at all uncomfortable—indicates little or no recruitment. Very little perstimulatory adaptation with R ear masked appropriately.

	250	500	1 kc.	2 kc.	4 kc.	8 kc.
L ac	40	60	65	65	100	..
				(bc data show loss, but exact figures are not possible)		
R ac	0	5	0	5	10	80

Conclusion thus far: Extreme conductive deafness from otitis media, with cochlear damage probably from bacteria, toxins, poisons entering through the round window.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/2 kc.	5.5 db.	300 msec.	(Control)
L/2 kc.	3	300	
L/500	2	100	

Here the relatively slight cochlear damage overrides the extreme peripheral loss to yield extremely flat integration slopes of 2-3 db.

This patient included to show the sensitivity of the slope of integration to a minor cochlear lesion.

PATIENT 8.—Age 23. Otoscopy negative. No family history or noise exposure. Recruitment at 500 after a delay of 20 db. Marked perstimulatory adaptation in R ear even as low as 500 cps.

	250	500	1 kc.	2 kc.	4 kc.	8 kc.
R ac	40	35	35	20	30	30
bc	5	—2.5	2.5	10	5	0
						("Carhart notch")
L ac	10	10	10	5	5	5

Conclusion thus far: Largely peripheral conductive loss, probably otosclerosis.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
L/500	12 db.	300 msec.	(Control)
R/500	13	100	

Perstimulatory adaptation indicates a minor nonconductive component; this tends to be confirmed by the integration data at R/500 where T_0 is rather considerably shortened over the control.

This patient included to show that the bc data, which seem to indicate a pure peripheral conductive lesion, can well be supplemented with additional information from integration data.

PATIENT 9.—Age 27. Defective hearing since childhood, etiology unknown. Radium therapy did not improve audiogram. Otoscopy negative. Recruitment absent at 250 and slight and long-delayed at 2 kc.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.
R ac	30	45	40	35	30	45	50	45
					(L ear entirely normal)			
R bc	—5	5	10	5	15			
					(L ear carefully masked)			

Conclusion thus far: Peripheral conductive lesion not accessible to otoscope. Slight high-frequency end-organ lesion.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
L/250	11 db.	200 msec.	(Control)
R/250	14	200	
R/500	7	200	
R/1 kc.	7.5	85	
R/2 kc.	5.5	150	

Data in defective ear at 500, 1 kc., and 2 kc. show abnormalities of slope and at 1 kc. in addition to a quite shortened T_0. This may mean that in this patient the integration data are very sensitive to the slight cochlear lesion (maximum of 10 db. as revealed by bc). This is in contradistinction to the data of Patient 23, where a conductive component completely overrode a heavy "perceptive" loss.

This patient included to show that the relative contributions of "conductive" and "perceptive" components cannot easily be assessed. In this patient, as in Patient 23, the picture is very complicated.

PATIENT 10.—Age 25. Diesel engineman for 10 years, history otherwise negative. Slight, straight-line recruitment.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.
R ac	—5	0	20	50	55	60	65	40
					(L ear entirely normal)			
bc	—2.5	2.5	7.5	beyond instrument				

Conclusion thus far: Monaural traumatic lesion of organ of Corti.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/500	13 db.	150 msec.	(Control)
L/1 kc.	9	150	(Control)
R/1 kc.	8	120	

Data from Miskolczy-Fodor and from others of our patients with traumatic lesions of the organ of Corti agree that such lesions are accompanied by recruitment and definite disturbance of the integration process. However, this patient's data are not significantly different from normal. Also, recruitment not quick.

This patient included to show how integration data can bear on the possibility that the defect in the R ear of this patient is

not of traumatic etiology, as would certainly otherwise be supposed.

PATIENT 11.—Age 30. High-frequency dip of long standing, etiology unknown. Otoscopy negative. Quick and complete recruitment, but not asymptotic type. No perstimulatory adaptation whatever.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.
L ac	—5	0	—5	0	50	50	30	10
	(R ear similar. All audiograms carefully studied in 1 db. steps)							
bc	—15	—15	—12.5	—7.5	30	30		—25

Conclusion thus far: Lesion of organ of Corti, not traumatic.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
L/1 kc.	4.5 db.	200 msec.	(Control)
L/2 kc.	11.5	140	(Control)
L/3 kc.	4	120	
L/4 kc.	4	140	

Data at one of the two control frequencies seemingly abnormal, with flatter slope than usual; but frequencies with audiometric loss both show even flatter slopes and tend to confirm the conclusion of an end-organ lesion.

This patient included to show how integration data agree with bc and the presence of recruitment to bear upon the locus of the lesion, and to show that disturbances of integration are not always accompanied by perstimulatory adaptation.

PATIENT 12.—Age 30. Hearing defective in grade school. Mumps, measles, diphtheria; bilateral mastoiditis at age 3. Fractured skull at age 8. Hot-shell man on 3-in. guns. Diesel engineman for 12 years. Has pretty good hearing, all things considered. Otoscopy negative, very slight recruitment.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.
R ac	35	40	35	50	70	95	90	80
						(R bc uncertain)		
L ac	0	5	10	15		55		40
bc	5	5	5	20		40		

Conclusion thus far: Bc and recruitment tests strongly suggest middle ear lesion undetectable by otoscopy, plus a relatively minor end-organ component increasing at high frequencies.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
L/250	10.5 db.	150 msec.	(Control)
R/250	6	150	
L/2 kc.	4.5	150	
R/2 kc.	4	150	

The stated "Conclusion thus far," above, developed in the absence of integration data, supposes a peripheral lesion undetectable by otoscopy. However, the integration data put an entirely different light on the matter: it appears that the lesion is within the cochlea and furthermore not on the organ of Corti. In R/250, the very minor flattening of slope is perhaps within normal limits.

This patient included to show the light thrown on the locus of lesion and on a difficult diagnosis by integration data.

PATIENT 13.—Age 59. Otoscopy negative. Somewhat variable low-frequency loss. Quick straight-line, not asymptotic recruitment, delayed 10 db., when 2600 c/s is balanced against 2 kc in L ear. (This is not a type of curve usually found in acoustic trauma or Ménière's disease.)

	250	500	1 kc.	2 kc.	2600	3 kc.	4 kc.	6 kc.	8 kc.
L ac	15	10	5	0	30	85	100	100	95
R ac	15	0	0	0	0	10	25	30	20

Conclusion thus far: High tone cochlear deafness.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/2600	8 db.	280 msec.	(Control)
L/1 kc.	7	200	(Control)
L/2600	4.5	500	

Much flatter slope in affected ear/frequency.

This patient included to indicate that cochlear lesions in addition to acoustic trauma and Ménière's disease can distort integration data.

PATIENT 14.—Age 40. Extremely variable loss at 250-2 kc.; stable loss at 4-8 kc. Explosion trauma in World War II; much tinnitus still. No perstimulatory adaptation 250-4 kc.; but at 8 kc. a tone 40 db. over threshold becomes inaudible within 40 sec. Recruitment at 1 kc. is delayed 10 db., then proceeds quickly to an asymptote 5 db. short of being complete (data taken 3/14/57).

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	8 kc.	
R ac	5	0	5	5		35	50	
R bc	0	0	—2.5	—5	25	..	..	
L ac	50	50	45	25		65	65	(3/4/57)
	20	15	20	10		65	55	(3/14/57, a. m.)
	20	20	15	10	10	65	55	(3/14/57, p. m.)
	40	35	30	10		60	65	(3/19/57)
L bc			25					(3/19/57)
L ac	20	10	10	0		65	55	(5/23/57)
L bc	5	7.5	10	0	27.5	..	..	(5/23/57)

Conclusion thus far: High-tone loss from noise trauma. Low-tone loss within the cochlea, but not on end-organ (note that bc varies with ac and that a non-end-organ type of recruitment exists). Cause unknown.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/1 kc.	8 db.	200 msec.	(Control)
L/1 kc.	8	140	

All integration data essentially normal.

This patient included to show that all cochlear defects yielding some types of recruitment do not always show disturbances of integration data.

PATIENT 15.—Age 42. Drum perforated at age 28 from otitis media. Origin of perceptive component unknown. Slight restriction of dynamic range (tested at 125 SPL at 1 kc. in free field) indicates some slight recruitment.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.	
R ac	45	45	45	50	45	55	30	35	(L ear similar)
bc	35	35	30	35	25	20	..	..	

Conclusion thus far: Otoscopy and lack of quick recruitment indicate a conductive impairment; bc strongly suggests an additional cochlear or neural lesion. No support for the latter.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/250	7 db.	200 msec.	(Control)
R/500	10	200	
R/1 kc.	7.5	150	
R/2 kc.	8.5	60	
R/3 kc.	12.5	75	

Essentially normal slopes; at 2-3 kc. T_0 is very brief. Normal integration data at low frequencies suggest that the predominantly "perceptive" loss (note the lowered bc) is in fact of central origin, or else that certain non-end-organ lesions within the cochlea are capable of supporting a normal integration process. At the higher frequencies, the shortened T_0 leads one to look for an additional defect within the cochlea.

This patient included to show how integration data can be used in an extremely difficult diagnostic case to rule out end-organ involvement where loudness-balancing is impossible.

PATIENT 16.—Age 53. Twenty years high-frequency tinnitus and hearing loss. Etiology unknown. Otoscopy negative. Only very slight recruitment at 3 kc.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.	
R ac	5	5	10	20	30	50	60	65	(L ear similar)
bc	—2.5	—2.5	—5	15	35	..	..	..	

Conclusion thus far: Slowly progressive cochlear or neural deafness, but if cochlear the lesion is not on the organ of Corti.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/1 kc.	10 db.	180 msec.	(Control)
R/2 kc.	3	200	
R/3 kc.	8	150	
R/4 kc.	10	75	

One of the frequencies (2 kc.) with "perceptive" hearing loss shows a much flatter slope than control, and one frequency (4 kc.) shows a much shortened T_0.

This patient included to show that, when there is no evidence of cranial nerve or C. N. S. damage, integration data can help to explore the possibility that conductive lesions exist within the cochlea.

PATIENT 17.—Age 15. Defect for at least five years, stabilized for at least the last three. Family history of allergy. At age 2-10 years, subject to severe "attacks," possibly parasitic hepatitis. Routine neurological examination negative. Otoscopy negative. Recruitment entirely absent.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.	
L ac	0	5	20	40	50	55	60	50	(R ear similar)
bc	—2.5	2.5	17.5	32.5	40	40	..	30	

Conclusion thus far: Lesion either neural or within the cochlea but not in the organ of Corti.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
L/250	7 db.	200 msec.	(Control)
L/500	8.5	200	
L/1 kc.	6.5	200	
L/2 kc.	9.5	150	
L/3 kc.	5.5	150	
L/4 kc.	4	400	

Two highest frequencies show flatter slopes than control; together with absence of recruitment, this tends to indicate a non-end-organ lesion within the cochlea.

This patient included to show that recruitment and integration distortion do not always coincide, and thus can work together to increase the precision of diagnosis.

PATIENT 18.—Age 47. Defect noticeable in past decade. Family history of deafness, maternal and paternal. Measles, mumps, scarlet fever. Otoscopy negative. Recruitment absent.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.	
R ac	0	10	25	25	30	25	40	25	(L ear slightly worse)
bc	0	12.5	22.5	27.5	30				

Conclusion thus far: Conductive lesion in cochlea, probably early familial presbycusis.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/250	13.5 db.	100 msec.	(Control)
R/500	11	90	
R/1 kc.	7.5	110	
R/2 kc.	5	100	

Slopes decrease regularly with higher frequencies.

This patient included as another almost crucial case on the question of the coexistence of recruitment and integration disturbance. Here, in presbycusis, which generally does not yield recruitment, we have a definite flattening of slope of integration. We conclude that integration must be looked on as a clinical sign in itself, and is not simply another test for recruitment. The power of its contribution is thereby increased.

PATIENT 19.—Age 51. Meningitis at 37, left side paralyzed. L ear almost totally deaf. Mother deaf at an early age. Concussion at age 20, but noticed no subsequent hearing loss. Otoscopy negative. Recruitment entirely absent.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.	
R ac	0	10	15	40	80	75	80	70	(L ear similar)
bc	2.5	12.5	15	40	below instrument				

Conclusion thus far: Lesion either conductive within cochlea, or neural; probably the former, from early familial presbycusis.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/250	9 db.	100 msec.	(Control)
R/500	8	130	
R/1 kc.	8.5	70	
R/2 kc.	6.2	80	

Another crucial case on the relation between recruitment and integration. Here, recruitment is entirely absent, yet at 2 kc. the slope is somewhat flatter than the control, and at 1-2 kc. the T_0 is quite short. If the two tests do not coincide, then the interest in integration data is increased, since it can yield new and perhaps vital information about the ear.

This patient included also to throw light on presbycusis. If our diagnosis of early familial presbycusis is correct, we have here one intracochlear condition which does not yield recruitment but does exhibit disturbed integration data.

PATIENT 20.—Age 35. Defect slowly increasing. "Carhart notch" at 2 kc. in bc. No perstimulatory adaptation at 2 kc., but noticeable at 3 kc. and very marked at 4 kc. (tone 25 db. over threshold becomes inaudible in 20 sec.)

	250	500	1 kc.	2 kc.	4 kc.	8 kc.	
R ac	45	50	55	45	45	45	(L ear similar)
bc	0	7.5	20	27.5	17.5	7.5	

Conclusion thus far: Otosclerosis, with additional high tone nonconductive component.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/250	10 db.	150 msec.	(Control)
R/2 kc.	7.5	200	
R/4 kc.	3	65	

Integration data agree with data from perstimulatory adaptation, to the effect that a "perceptive" component enters at the high frequencies.

This patient included to show how integration data can help to delineate the frequency region at which a high-tone nonconductive component begins.

PATIENT 21.—Age 19. L eardrum thickened, lusterless, retracted. Slight scarring. Handle of the malleus pulled backward into the posterior quadrant. X-rays on L mastoid show evidence of old extensive otitis media, with marked sclerosis in the mastoid areas leaving few or no air cells remaining. Slow straight-line recruitment.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.	
L ac	35	40	40	20	15	30	35	25	(R ear entirely normal)
bc	7.5	2.5	5	5		10		0	

Conclusion thus far: Pure conductive deafness from past otitis media.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
R/1 kc.	14.2 db.	200 msec.	(Control)
L/1 kc.	4.8	120	

The slope of 4.8 is much flatter than that of the control.

This patient included to show how integration data emphasize the fact that some recruitment was present, even though the bc data and the otologist's diagnosis point to a pure conductive loss which should have no effect on recruitment or integration. It may be that a slight cochlear loss exists also, which would otherwise be overlooked.

PATIENT 22.—Age 25. Otoscopy negative. No family history of serious diseases. As a child, hearing would "weaken" when he had the usual colds, but no discharge or pain is recalled. Noticed trouble about six months after starting work as a ship's boilerman four years ago. Worked there for two years. Occasionally he was on duty for as long as 12 hours at a stretch. Very slight recruitment at 250 (ac balanced against bc, L ear); fast recruitment at 1 kc. (L ac at 1 kc. balanced against L bc at 250).

	250	500	1 kc.	2 kc.	4 kc.	8 kc.	
L ac	35	40	45	25	30	10	(R ear similar)
bc	0	15	35	30	15	—5	

Conclusion thus far: Preponderantly conductive peripheral loss, probably from old subclinical otitis media; overlaid more recently by an end-organ dip centered at 1-2 kc., probably from ship noise (Note: there is some reason to believe that ears with a history of otitis media, even subclinical, are more susceptible to damage from noise.)

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	T_0	
L/250 bc	9 db.	500 msec.	(Control)
L/1 kc. ac	6	130	

Integration data at 1 kc. (slightly flatter slope, shorter T_0 than control) confirm the conclusion from bc and recruitment data that an end-organ lesion overlies the peripheral conductive loss.

This patient included to show how integration data can help confirm the real nature

of the bc dip—it is probably not the "Carhart notch"—and thus help to rule out otosclerosis.

PATIENT 23.—Age 40. Otomycosis and otitis externa. High-frequency defect from gunfire. Recruitment delayed but complete.

	250	500	1 kc.	2 kc.	3 kc.	4 kc.	6 kc.	8 kc.
R ac	0	20	35	40	60	80	80 (10/31/55)	65 (10/31/55)
bc	0	0	5	5	20	25	..	..
R ac	0	0	5	0	35 (6/5/56)	35	60 (Left ear same)	45

Conclusion thus far: Temporary conductive impairment largely cleared up after six months. Permanent high-tone traumatic dip.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	To	
R/250	10 db.	200 msec.	(Control) 10/31/55
R/500	9	200	10/31/55
R/1 kc.	10	200	10/31/55
R/3 kc.	8	200	10/31/55
R/3 kc. (after recovery)	3.2	350	6/5/56
R/4 kc.	3	110	6/5/56

Integration data distorted by acoustic trauma, but not until a conductive peripheral impairment had been removed.

This patient included to point out that we cannot always confirm Miskolczy-Fodor's assumption that integration data will always allow one to separate the amount of contribution of the conductive and the "perceptive" components.

PATIENT 24.—Age 20. Otoscopy negative. Exposed to high noise levels both continuous and intermittent in military service. No perstimulatory adaptation whatsoever at 1 kc., but marked at 3 kc. and higher.

	250	500	1 kc.	2 kc.	4 kc.	8 kc.
L ac	25	25	25	20	80	80
bc	20	20	20	20	..	..
R ac	..	..	100	100	..	..

Conclusion thus far: Pure cochlear or central deafness (bc=ac).

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	To
L/1 kc.	4.2 db.	200 msec.

No control ear/frequency, but data definitely not normal.

This patient included to show that integration data is in some cases more sensitive to "perceptive" deafness than is perstimulatory adaptation, especially at the lower frequencies.

PATIENT 25.—Age 22. Otoscopy essentially negative. Maximum PB speech reception: 66%. No perstimulatory adaptation.

	250	500	1 kc.	2 kc.	4 kc.	8 kc.
R ac	30	35	35	40	50	60
bc	20	27.5	27.5	40	45	..
L ac	45	60	55	60	65	50

Conclusion thus far: Pure nonperipheral defect of long standing, of a type to garble speech; could be cochlear or central. Cause unknown.

Summary of Integration Data and Their Contribution

Ear/Frequency	Slope of Integration	To
L/500	16 db.	300 msec.
R/1 kc.	8	200

This patient included to show that the combination of garbled speech plus no disturbance of integration would lead one to suspect a C. N. S. involvement rather than a cochlear lesion.

Comment

We agree with Miskolczy-Fodor that traumatic lesions of the organ of Corti invariably produce atypical integration data, and that the slope of integration is proportional to the extent of the lesion. Furthermore, it is likely that cases of Ménière's disease, streptomycin damage, and certain other disorders of the organ of Corti would show the same sort of relation between audiometric loss and slope of integration.

We do *not* find, however, that all cases of intracochlear disorders yield atypical integration data. Nor can we substantiate Miskolczy-Fodor's contention that in all cases of mixed deafness it is possible to assess the extent of the "perceptive" component by noting the slope of integration. We see in Patient 23, at R/3 kc., with a 60 db. loss, of which 35 db. is definitely known to be perceptive, that quite normal integration data are yielded. Only when the conductive component cleared up did the perceptive component produce the characteristic flatter slope. Another example, Patient 9, at R/2 kc., shows a characteristic flatter slope even though the perceptive component can amount to only 5 db. of the 35 db. total hearing loss.

It seems a reasonable assumption that nerve or brain lesions will not of themselves render the integration process *more* efficient, and we definitely know that conductive lesions in the tympanum do not do so. If this is correct, we may then have, in integra-

tion data, a potentially valuable tool in diagnosis. We lay considerable stress upon the fact that in many of our patients there was recruitment but no disturbance of integration, while in others there was no recruitment but a definite disturbance of the integration process. It seems to us that in the long run such a situation is clinically much more valuable than that the two should always coincide. Since *additional* information can be added by an integration test; the nosological search can to an extent be guided and sharpened.

We can say very little at this time about the sorts of intracochlear lesion which yield atypical integration data. We know that presbycusis does do so in the absence of recruitment (Patients 18, 19), and that some cochlear disorders other than presbycusis do so also in the absence of recruitment (7, 17). We know that acoustic trauma does so accompanied by quick, asymptotic recruitment (Patients 3, 4, 5, 6, 22, 23). We know that at least one type of cochlear disorder does so, accompanied by quick but straight-line (not asymptotic) recruitment (11, 13), and that some types of cochlear disorder do so, accompanied by slight straight-line recruitment (8, 9, 12, 16, 21).

We can, on the other hand, say even less about the sorts of intracochlear lesion which yield *normal* integrative data. We present one such case (14) exhibiting quick, asymptotic recruitment, and two cases (10, 15) yielding slight, straight-line recruitment.

Presumably, as such data mount, together with more and more precise diagnosis, the future contribution of such audiological data will increase. As yet we are only in the pioneer phases of such a goal. Perhaps lesions producing added stiffness, mass, or friction can be induced in the experimental animal. Certainly selective destruction of the organ of Corti is possible. We, and we hope many others, will be exploring these areas in the future.

Of interest is the question whether Miskolczy-Fodor's techniques and methods of reporting data are optimum for the situation. Let us first take a look at his fundamental equation for energy integration in the normal ear:

$$N=9 \log \frac{T}{T_0=150}$$

Where

N=threshold shift for a tone of duration T, as compared with threshold for a tone of duration T_0 (that duration, in milliseconds, below which energy integration is linear). However, most previous papers treating this equation report a constant of 10 instead of 9, and a T_0 nearer 200 than 150.

Let us examine how changes in these constants affect the clinical picture.

A convenient first step is to redraw Miskolczy-Fodor's [9] Figure 4, which is based on the constants 9 and 150. Figure 1 presents the curve from his Figure 4, plus curves using the constant 10 instead of 9, and, in succession, T_0 of 75, 150, 100, and 200 msec.

It is seen that adopting the constant 10, indicating perfect temporal integration for

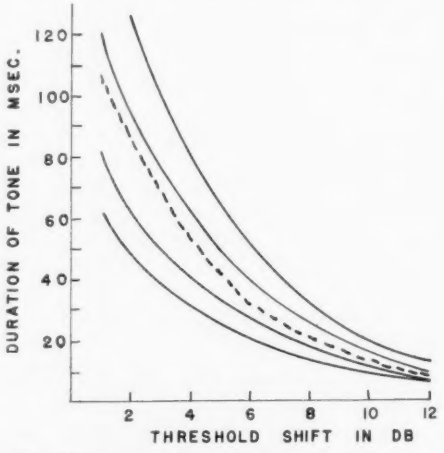

Fig. 1.—Amount by which the intensity of a brief tone must be increased to reach threshold, as compared with intensity at threshold of a tone of infinite duration. Note: All curves drawn from the equation: Threshold shift in db.$=K \log \frac{T}{T_0}$ where K is a constant, T_0 is the critical duration (see text) and T is tone duration in msec. (From Miskolczy-Fodor,[9] Fig. 4).

Curve (left to right)	K	T_0
—	9	150
(*a*)	10	75
(*b*)	10	100
(*c*)	10	150
(*d*)	10	200

the normal ear, has the effect of changing the time threshold by only 2-10 msec. for any threshold shift, the weaker threshold shifts being more affected. This slight difference is well within experimental error, and we conclude that no real discrepancy will result if we raise the constant 9 up to 10, to fit our own and some other data a little better.

Figure 1 shows, however, that adopting a different T_0 than 150 msec. will change the clinical picture by a considerable amount, and that this amount will be directly proportional to the T_0 chosen. For example, the curve for $T_0=200$ can be derived simply by multiplying that for $T_0=150$ by 1.33. Similarly one can multiply by 0.67 to derive the curve for $T_0=100$. This follows, of course, from the form of the equation for these curves, but it may be easier to grasp visually from Figure 1. For example, at 3 db. sensation level with $T_0=150$, the time threshold is 75 msec.; but with $T_0=200$, the time threshold is $1.33\times75=100$ msec.

Thus, it matters which T_0 is used as the normal case in the integration equation. Any further calculations for a defective ear will depend directly on the choice of T_0 for the normal case.

Miskolczy-Fodor, as we have seen, settled upon $T_0=150$ msec. For clinical use with a partially deafened ear, he first finds threshold with a T_0 of 150 msec., then increases the intensity by, say, 3 db. and finds a time threshold in milliseconds for the 3 db. sensation level. He then looks up the *normal* threshold shift for that particular time threshold by using his Figure 4 (our Fig. 1, Curve *d*). Finally, to express the increased loudness of the patient for that particular time threshold, he divides the *normal* threshold shift by the *patient's* threshold shift. This is his loudness improvement ratio. *This,* if a patient has to turn the intensity up 3 db. in order to hear a tone of 15 msec. duration, whereas a normal ear has to have a 10 db. shift, the patient is said to have a loudness improvement ratio of $\frac{10}{3}=3.3$.

Now if 150 msec. is in reality the normal T_0 *for that patient,* and his pathological T_0 is also 150 msec., then the loudness improvement ratio will be constant for any and all threshold shifts used in calculating the ratio, and will be an excellent way to describe the data.

(A corollary is that if T_0 and T_0 are equal or nearly so, for the normal case and for the pathological case also, then the loudness improvement ratio and the slope of integration are equally good for describing the data. The two indexes are in fact mathematically related according to the curve in Figure 2. The shape of this curve is entirely independent of the T_0 chosen, except that the relevant curve in Figure 1 should be used to calculate the loudness improvement ratio of Figure 2. Figure 2 states that a loudness improvement ratio of 2.0 occurs with a slope of integration of 5 db/1 log unit duration; a loudness improvement ratio of 2.5 occurs with a slope of 3.8 db., and so on.

But, unfortunately, before Miskolczy-Fodor's loudness improvement ratio can be

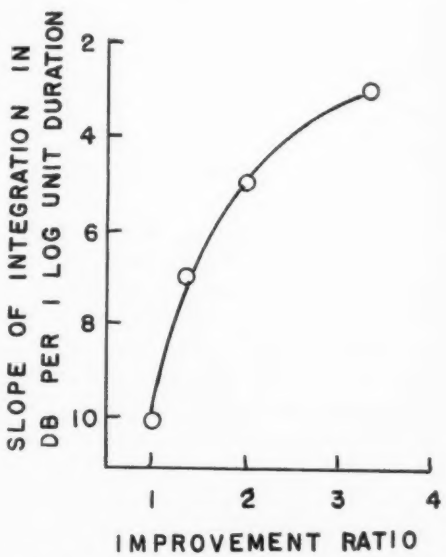

Fig. 2.—Showing that the loudness improvement ratio is related to the slope of integration as above, no matter what value is chosen as T_0, provided only that the patient's T_0 and the value chosen as the normal T_0 are similar.

universally adopted, one must wrestle with three questions: (*a*) whether the pathological ear always yields a linear slope of energy integration; (*b*) whether $T_0=150$ msec. is a sufficiently close approximation to the normal T_0, and (*c*) whether T_0 changes in the pathological ear.

(*a*) *Does the pathological ear always yield a linear plot of threshold-shift vs. tone duration?* Our experience has been that the statement is generally correct. In several patients we have taken the opportunity to define carefully the slope of integration, using a half-dozen or more tone durations; usually we find that within the range of variability a straight line is the best fit. There are a few real exceptions. And there may of course be a slight rounding where the line intercepts zero threshold shift. But we feel that Miskolczy-Fodor's assumption in this matter is fairly well justified.

(*b*) *Is it possible to fix upon 150 msec. as the normal?* Our data on a half-dozen normal-hearing individuals (12 ears) show that in a perfectly normal ear T_0 may vary from 100-300 msec., and in this we confirm the data in Miskolczy-Fodor's Table I, where it can be calculated that in about $\frac{1}{6}$ of all normal ears T_0 would be about 100 msec. or less, and in about $\frac{1}{6}$ it would be about 230 msec. or more. Evidently 150 msec. is not always the best estimate of normal T_0 for a particular patient.

Let us now consider the effect of using the "wrong" normal T_0 on the constancy of the loudness improvement ratio. Let us assume that the patient's control or "true" T_0 is *less* than 150 msec.; then, if one uses the 150 msec. curve in Figure 1, the loudness improvement ratio will seem to grow smaller and smaller as one calculates it at 2, 4, 6, etc., db. threshold shift; and conversely it will seem to grow larger and larger with threshold shift if the normal T_0 chosen is *larger* than 150 msec. And the greater the discrepancies between the true T_0 and 150 msec., the greater will be the discrepancies in the ratios.

We feel that it is not possible to settle upon any single T_0 as the normal case, and that wherever possible a T_0 should be sought *in the patient* to use as the best estimate of the normal T_0 for him. Where no normal or nearly normal ear/frequency exists, the best compromise seems to be to use $T_0=200$ msec., according to our normative data.

(*c*) *Does T_0 change in the pathological ear?* Very often T_0 decreases drastically whether one considers a normal ear/frequency in the patient, or the range of normal-hearing ears. Often it is, indeed, this shift in T_0 rather than a change in slope of integration which characterizes an ear. With Patient 15, for example, the slopes of integration at the highest frequencies is well within normal limits, but the T_0 has been reduced to 60 and 75 msec. Other examples can be found in our files (see Patient 16, at R/4 kc.). In such cases, it is hard to believe that it is quite correct to apply a formula based upon $T_0=150$ msec.

In our series we have come upon over a dozen ears where T_0 was shortened to less than 100 msec. In these instances we feel some device other than the loudness improvement ratio as calculated by Miskolczy-Fodor should be sought.

(*d*) *How then should integration data be collected and reported?* Because of the importance as outlined above of determining as accurately as possible the "normal" T_0 for any patient, we would recommend a procedure which does just that as a first step, then determines T_0 and the slope of integration for the defective ear/frequency under consideration. Anything less than this is a compromise using assumptions which may not be justified in a particular patient.

Specifically, our procedure is as follows:

1. Find if possible some frequency on at least one ear for which the audiogram is or approaches normal. Determine the normal or control T_0 by taking threshold with a tone duration of 300 msec. Then, with successive steps of 200, 150, 100, 75 msec., or intermediate steps as the data indicate, find

the duration below which threshold shifts. This is T_0. Now reduce the duration by 1 log unit (this would be $\frac{1}{10}$ T_0) and determine threshold shift. The shift in threshold between the duration T_0 and $\frac{1}{10}$ T_0 is the slope of integration in decibels. It is wise to find threshold shift at two or three intermediate points to determine more precisely the slope.

Now find T_0 and the slope of integration for the ear/frequency on which integration data are desired.

3. If the normal T_0 and the pathological T_0 the the same, or within perhaps 25 msec. of each other, it is completely justified to calculate Miskolczy-Fodor's very handy loudness improvement ratio. Do this by entering our Figure 2 with the slope of the pathological ear, or by way of the appropriate curve in our Figure 1 as explained earlier.

4. If the pathological T_0 is much shortened, then the ratio is not really applicable. One should report both the pathological slope and some index of the relation between the normal and the pathological T_0. The necessity here of reporting more than the ratio is made clear by our Figure 3, in which it is seen that the ratio is related in no simple way to the relation between the normal and the pathological T_0. This means that the improvement ratio cannot directly contain the added information provided by a shortened T_0. For example, with Patient 16, at R/4 kc., the slope of integration is 10, which looks normal, and the improvement ratio at 6 db. threshold shift is only 1.4. So far we would suppose that R/4 kc is only slightly atypical; but as a matter of fact the T_0 has been shortened from a control of 180 msec. to 75 msec., and the true loudness improvement ratio is certainly about $\frac{180}{75}=2.4$. This follows from Figure 3, from which it can be shown that the average improvement ratio is about equal to the ratio of normal T_0 to pathological T_0. (Read Fig. 3 vertically at any T value to confirm this general conclusion.) For example, if a patient shows a slope of 10 and a T_0 of 75 msec. for a particular ear/frequency, and his control T_0 is 150, then Figure 3 states that his average improvement ratio can be found by adding up and averaging all the improvement ratios calculated by using the threshold shifts 1, 2, 3 . . . . 10; or, it can be more easily approximated by simply dividing $\frac{150}{75}=20$. (For a different normal T_0 and a different slope, Figure 3 will of

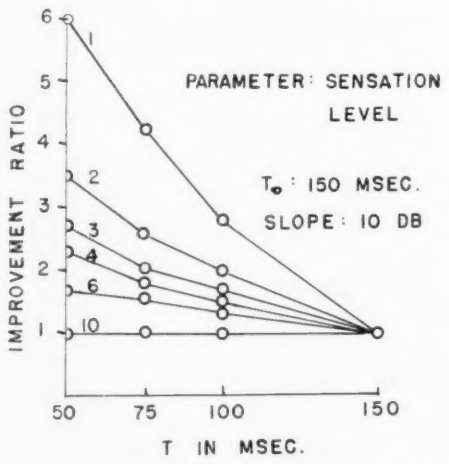

Fig. 3.—Improvement ratio as a function of T, for several sensation levels, calculated according to the formula:

Improvement ratio=10 log $\frac{T}{T_0}$ (150 msec.)

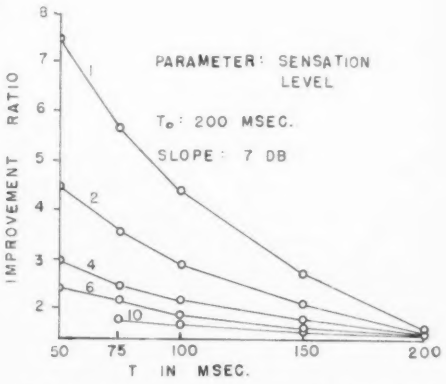

Fig. 4.—Improvement ratio as a function of T, for several sensation levels, calculated according to the formula:

Improvement ratio=7 log $\frac{T}{T_0}$ (200 msec.)

course not exactly apply. Figure 4 gives the case for a normal T_0 of 200 msec. and a slope of 7 db. A fair approximation to a loudness improvement ratio, averaged for all threshold shifts, is still found by using the generalized formula:

$$\text{Improvement index} = \left| \frac{T_0 \text{ (Normal)}}{T_0 \text{ (Pathological ear/frequency)}} \right|$$

In case a normal T_0 cannot be determined at any ear/frequency, a control T_0 of 200 msec. may be used without great distortion.

In this way, when Miskolczy-Fodor's ratio cannot properly be applied, we salvage an improvement index from the data, and retain the very useful advantage of compressing the data into a single number which compares the defective ear with the normal condition.

Summarizing this section, we can point out that there are three solutions, or progressive compromises, which can be adopted:

1. If the control and pathological T_0's are both 150 msec., use Miskolczy-Fodor's loudness improvement ratio.

2. If the two T_0's are similar, but differ from 150 msec., then calculate the loudness improvement ratio, but from the appropriate curve in our Figure 1 rather than according to the procedure of Miskolczy-Fodor.

3. Where the pathological T_0 is so short that neither Miskolczy-Fodor's ratio nor our modification using the patient as his own control can serve, we recommend reporting simply the slope and T_0. If for clinical reasons it is desired to relate the ear to the normal case by using a single comparative number, use the improvement index.

It seems to us that at this stage little is gained by using comparative numbers; we feel that more will be accomplished in thinking about how the ear stores and utilizes energy over time if one thinks in terms of the slope of integration. For example, consider patients who can hear tones of 20 msec. duration with a relative improvement of 6-7 db. over the normal ear. What we wish to know with these patients, from the scientific point of view, is not whether they are 1.5 or 2.3 better than normal. What we really wish to know is how the defective ear can summon up additional fibers to subserve threshold relatively far better than the normal ear. It would seem more fruitful to seek how, for example, a difference in effective band width may lie at the basis of differences between normal vs. pathological slopes, rather than to stop at a simple numerical comparison.

Summary and Conclusions

A test was made of Miskolczy-Fodor's important thought that the way a partially deafened ear integrates energy over a duration of time may provide a clue to the locus of the disorder.

In the normal ear, a shift in threshold of 9-10 db. is found when stimulus duration is shortened 1 log unit (below some critical duration T_0.) However, in some partially deafened ears the threshold shift may amount to only 3 db. It was assumed by Miskolczy-Fodor that a reduction in threshold-shift below the 9-10 db. norm was related to end-organ pathology, and was coincident with the presence of recruitment.

A selected series of 25 defective ears is presented, using the slope of integration and the T_0, which compresses into a few numbers a great deal of information as to the integrative power of an ear.

We were unable to confirm Miskolczy-Fodor's opinion that integration data will in all patients allow one to separate "conductive" and "perceptive" components, or that atypical integration data always and only accompanied recruitment. We found some types of intracochlear disorder exhibiting both, some types neither, some types either one alone.

This lack of correspondence between integration data and recruitment, far from detracting from the usefulness of the Miskolczy-Fodor test, makes it much more valuable potentially than would otherwise be the case. It means that a significant amount of additional information is brought to bear upon the disorder. By analyzing the profile of test results which a defective ear may

exhibit in audiometry, recruitment, integration, perstimulatory adaptation, speech reception, etc., etc., one should eventually be able to fix the locus of lesion as definitely in the tympanum, within the cochlea, or definitely central to the cochlea; and it now appears that it is not too much to expect that particular test profiles may be exhibited by particular intracochlear disorders, thus increasing significantly the power of diagnosis. We have evidence that this is true for at least three intracochlear disorders.

We conclude that the Miskolczy-Fodor test can have a very real place in a completely equipped diagnostic clinic, and we trust that mounting data from this and other laboratories will eventuate in a clear picture.

U. S. Naval Medical Research Laboratory.

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Clinical Notes, New Instruments and Techniques

Tray for Bedside Tracheotomy Care

JAMES A. HARRILL, M.D., Winston-Salem, N. C.

The tray shown in Figure 1 for bedside use in tracheotomized patients has been useful in standardizing and simplifying the care of these patients.

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Division of Surgery, Department of Otolaryngology, Bowman Gray School of Medicine of Wake Forest College and the North Carolina Baptist Hospital.

The component stainless steel parts are from standard hospital stock-room supplies, except for the top, which is cut from a flat stainless steel sheet. Holes are cut to fit *A*, *B*, and *C* in Figure 2. The edges of the top are rolled to fit over tray *D*.

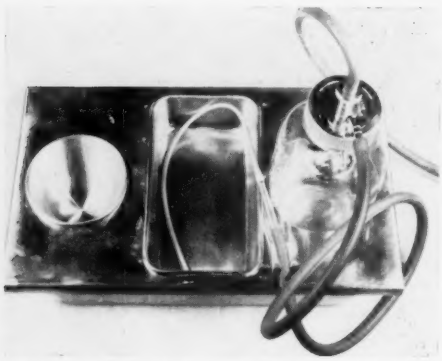

Fig. 1.—Assembled tray for bedside tracheotomy care. Size: 17×11×4 in.

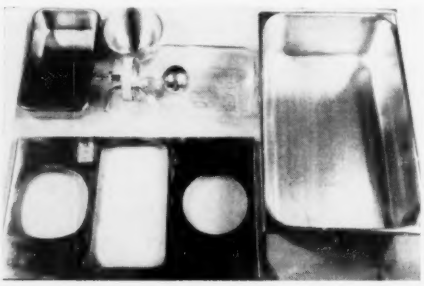

Fig. 2.—Component parts of tray: *A*, stainless steel catheter tray, size 9×5×2 in. *B*, stainless steel iodine cup, size 4½×2⅝ in. *C*, vacuum bottle with automatic float cut-off. *D*, stainless steel tray, size 17×11×4 in. *E*, top, cut from sheet of stainless steel.

Air-Healing Method for the Donor Site of Skin Grafting

E. E. MIHALYKA, M.D., and V. M. JORDAN, M.D., Cleveland

In otological work there have been many ways in which skin grafting has been advised and used, and this paper proposes to introduce another method in treating the donor site in skin grafting. The average hospital stay relative to skin grafting can be reduced almost 50%. The skin grafts, which have been used in connection with radical or modified endaural mastoidectomies, have been taken from the anterior portion of the thigh, usually with the Brown electric dermatome, although the Padgett dermatome may be used or free-hand grafts. A setting of 18 is used for this particular type of skin graft on the Brown dermatome. The anterior portion of the thigh is shaved first and then properly prepared with hexachlorophene (pHisoHex) and draped. A very thin coat of liquid petrolatum (mineral oil) is applied to the surface of the area of the graft. With adequate pressure the Brown dermatome is placed on the graft site area, and with the assistant putting tension on the skin with a tongue depressor, pulling in front of the Brown dermatome, a piece of skin of the desired length and width is taken. Thrombin, 1000 units, mixed with 10 cc. of isotonic saline, is withdrawn in a 20 cc. syringe and is then immediately placed over the donor site area (Figure, right). The thrombin

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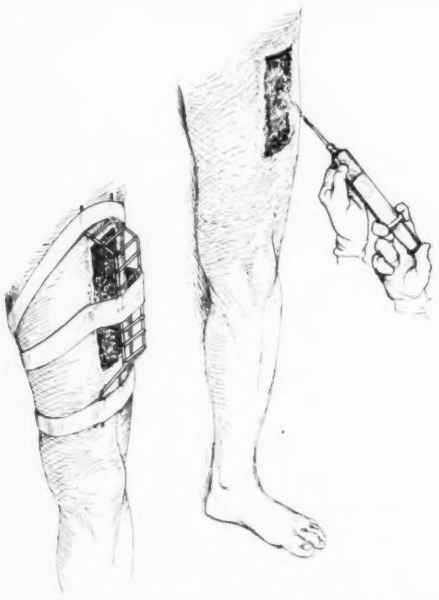
Clinical Instructor in Otolaryngology (Dr. Mihalyka); Clinical Professor of Otolaryngology (Dr. Jordan), Western Reserve University School of Medicine.



Right, thrombin applied over donor site area. Left, wire splint placed over donor site area.

should flood the whole area, and with a towel the donor site area should be fanned to help the clotting of the donor site. An orthopedic wire splint (Figure, left) is then placed over the donor site area and secured with adhesive tape in the anterior and posterior portions, with a large piece of tape across the center portion to prevent the sheet from falling against the donor site. Almost immediately epithelialization starts, and by the time the patient is taken back from the operating room to his ward a cradle is placed over the thigh to allow freedom for the patient's movement without injuring the donor site. The area starts to crust in about 24 hours, and in 48 hours the cradle is no longer needed. The donor site is for all intents and purposes completely healed in about five days. It has been found that this method is superior to the previous methods which we used in the following ways: 1. The time of healing of the donor site is cut down to approximately 50%. 2. The discomfort caused by pulling off the sterile petrolatum dressing every four or five days plus the pressure dressings over the donor site is nonexistent. 3. The donor site begins to heal almost immediately after the removal of the skin graft, whereas with the other method the healing is disturbed each time the sterile petrolatum dressing is removed and reapplied to the donor site area. 4. No untoward reactions have been noticed to date with this method. 5. Nursing care is minimized for the patient. Our series consists of more than 100 cases. The age range varied from 14 years through 61 years of age. Both sexes were involved. This method has also been used in dermabrasion in facial plastic work, as well as other plastic work.

Summary

A new method of air healing for donor site areas is presented. Healing time is reduced 50%. This new method is much more comfortable for the patient. Nursing time is reduced.

1726 W. Schaaf Rd. (9).

Progress in Otolaryngology

Tumors of the Nose and Throat

Summaries of the Bibliographic Material Available for the Years 1953 to 1956

KENNETH D. DEVINE, M.D., Rochester, Minn.

Changing concepts of the nature of cancer are altering the role of the surgeon in its management and are increasing his responsibility with reference to the long-range care of the patient. The surgeon, if he is interested in the management of cancer (and if he is not, he should not attempt the mere amputation of an organ) is the key person in the management of the disease. The days of carrying out "routine operation" have gone. There can be no "routine operation" because of the complex nature of the disease and the fact that a human being as well as a neoplastic process is involved. Dunphy [1] stated that even finally when the course of the patient is clearly downhill, the surgeon has a duty to remain in the picture to integrate the efforts of all concerned with the management of the patient. To be abandoned is worse than to die.

Prolongation of life is not the only aim of treatment: it should be a tolerable life. According to Asher [2] it is better to be wholly alive for one month than half alive for two months. It is not always worth the discomfort of major surgical procedures to get minor recovery.

Resection producing a monstrosity is of doubtful value. Operations that become heroic procedures and that leave appalling defects are in the nature of surgical stunts unless a cure or a long interval free from recurrence can be guaranteed. According to Moore and Faulkner [3] it is wrong to dabble and treat occasional patients without the necessary neoplastic background.

Cameron [4] estimated that about 19,500 new cases of cancer of the buccal cavity and pharynx occur annually in the United States; 6000 cases of cancer of the esophagus among men each year, and the same for the larynx. In 1953, 1800 men died from primary cancer of the larynx in the United States.

Analysis of vital statistics data by Phillips and Owchar [5] showed that a significant decrease in the mortality from cancer of the buccal cavity and pharynx in Canada between the years 1941 and 1953 (34.7% drop). Analysis of death rates attributed to cancer of the larynx, trachea, bronchus, and lung showed a significant increase in males (141.7%).

Shapiro and associates [6] noted that in the African the incidence of cancer of the maxillary antrum is remarkably high, and lung cancer is relatively rare. The exceptionally high incidence of carcinoma of the paranasal sinuses seems to be a unique feature characteristic of the South African Bantu, and presumably is dependent on local conditions. A prolonged and heavy intake of snuff was thought to play some part in the high incidence of the disease in the Bantu. Further inquiries by Keen [7] disclosed that the native adds to his snuff various, previously incinerated, herbal mixtures. These additives contain carcinogenic agents more

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Section of Plastic Surgery, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

potent than those in condensates of cigarette smoke.

According to an editorial in the *New England Journal of Medicine*,[8] the record of the medical profession in the care of the patient with hopeless cancer is not an enviable one. The treatment of advanced cancer is not a pleasant task. It inevitably has a grim and bitter side, and requires not only skillful manipulations of all available techniques but also extraordinary courage and equanimity on the part of both physician and patient.

Malignant lesions of the ear, nose, and throat in older people create many problems, and Brewer[9] noted that a group opinion is frequently welcomed by both the patient and his family. The physician then is released from having sole responsibility for a decision which he often finds himself loath to assume, both by virtue of training and by preference.

Brewer,[10] too, advocated group effort in treating patients with tumors about the head and neck.

Fitz-Hugh and Hobart[11] stated that palliation and pessimism concerning treatment of squamous-cell carcinoma of the upper part of the respiratory tract are yielding to improved curative measures and optimism. This has been accomplished by the ability to perform more extensive operations in the attempted extirpation of the lesion.

Martin and associates[12] asked, "Actually, how much basic difference is there between the custom of 'desertion' of the 'old' as practiced by primitive peoples such as the Indian, Lapp or Hottentot and the policy of withholding indicated surgery from the 'old' cancer patient?" To them the main significance of chronologic old age lies in the viewpoint of the person who employs the term. The preoperative examination of a patient at any age should be pursued for one purpose only, that is, to determine when an indicated operation had best be performed and not to establish whether it should be performed at all. When the operation promises a reasonable chance of cure or long-term palliation, the lesser evil surely is to face the risk of immediate mortality rather than to await inevitable death from cancer or to accept passively the increasingly intractable pain from the complications of the advancing cancer itself. Persons in the older age group have the same desire for, and the same right to, survival as those in the younger age group, and, furthermore, the great majority will be found to possess sufficient fortitude to face the operative hazard.

Too often physicians have applied the fact of the eventual hopelessness of incurable cancer to all aspects of the patient's care. Slaughter, Southwick, and Harris[13] stated that the result is neglect of the patient and dereliction of duty by physicians. Control of pain due to cancer may be achieved in two ways: (1) treatment which eliminates the tumor or causes temporary regression, and (2) treatment which ablates sensory perception of pain or eliminates specific pain pathways.

Head and Neck in General

Conway[14] makes a plea for consideration, in advance of excisional procedures, of the techniques of reconstructive surgery in the management of carcinoma of the oral cavity with the goal of improved results on follow-up.

Longacre and associates[15] recommend meticulous radical surgical ablation controlled by microscopic examination of frozen sections in most cases of cancer of the head and neck. Reconstruction, too, is emphasized.

The treatment of tumors of the head and neck requires a variety of skills which today are used in several specialties. For this reason these lesions are in a medical no-man's land. On the fringe of those toying with the problem are the

oral surgeons and oral pathologists, according to Fahey.[16]

Diagnosis.—Boyle [17] maintained that in most instances removal of a specimen for biopsy when the clinical impression is squamous-cell carcinoma should be performed by a specialist who by training and experience is best qualified to do so, and who will assume responsibility for the treatment.

A study of 370 patients with tumors of the head and neck seen at the Lahey Clinic by King [18] revealed that the commonest causes of missed diagnoses were failure to make a competent examination of the internal epithelium of the head and neck; misinterpretation of cervical lymphadenopathy; lack of awareness of the common occurrence of a second primary lesion; persistent and metastatic disease; confusion of lymphoma with infection, and undue reliance on negative reports of biopsy.

In the presence of general adenopathy the most desirable site of biopsy is usually the cervical region, according to Dawe.[19] It is important that the pathologist receive the excised node or nodes as nearly intact and untraumatized as possible. A fragmented specimen loses much of its value. A place exists for examination of fresh frozen sections of lymph nodes if the limitations of the method are recognized. It is often possible by this method to determine the adequacy or significance of the specimen. A fresh frozen section may offer guidance as to bacteriologic handling of the specimen. A good routine practice is to divide all specimens of lymph nodes into two portions, one for bacteriologic examination and one for histologic study.

If a definite diagnosis cannot be made by aspiration biopsy, a specimen should be obtained by formal biopsy. James [20] was of the opinion that it should be obtained in the operating room. Frozen sections should be made, and, if a diagnosis of carcinoma is made, the surgeon should proceed immediately with the surgical resection.

Krementz,[21] in a good paper, discussed the use and misuse of needle biopsy. Needle biopsy is a useful aid in the diagnosis of malignant lesions. Since spread may occur along the needle tract, the procedure may be elected in cases in which potential seeding can be controlled by excision, radiation, or systemic therapy. In cases in which seeding will not affect the patient more adversely than his disease, needle biopsy may be used to establish a tissue diagnosis.

Engell [22] studied the peripheral and venous blood draining the tumor area at the time of operation for rectal and lung cancers. The finding of cancer cells in the circulating blood confirms previous observations that the tendency to venous invasion and consequently to hematogenous spread of cancer cells depends chiefly on the grade of differentiation of the tumor. Presumably a venous spread of tumor cells takes place from all carcinomas of Grades 3 and 4. It was concluded that in some cases all the cancer cells entering the blood stream are destroyed. There was no evidence of a definite increase in the escape of tumor cells into the blood stream following operative manipulation. Ligation of all the veins near the tumor before the tumor is touched, therefore, would hardly reduce the incidence of hematogenous metastasis.

Treatment of Cancer.—All surgeons interested in the treatment of cancer should read an editorial on "The Case of the Cobalt 60 Unit" by Watson.[23]

In a particularly concise review El Sebai [24] gave the reasons for the modern trend in the treatment of cancer of the head and neck.

Coyle and Balofsky [25] presented treatment plans for handling cancer in the head and neck. The basic reasons for selection of such techniques are included.

They emphasize cooperation between radiologists and surgeons.

Copeland [26] outlined his method of treating common cancers about the head and neck with or without regional metastasis. There is a good bibliography of the pertinent literature, and the paper should be read by all interested in the subject.

Ochsner,[27] too, presented his opinions and reviewed some of the literature that dealt with cancer of the head and neck.

Murphy and Tonning [28] stated that there should never be a clinic rule for the treatment of any specific type of malignant oral lesion. The treatment of cancer of the mouth in their opinion, however, should be surgical in the main.

Probably in no other structure of the body is the necessity for close cooperation between the physician and radiotherapist more imperative than in the treatment of cancer of the head and neck. In the mounting enthusiasm for radical surgical procedures in this region, Coyle [29] considered that the equally remarkable achievements of the radiotherapist with his newer techniques have tended to be overlooked. A review of the fundamental principles of irradiation has been undertaken, together with the concept of biological radiosensitivity.

Sinclair [30] emphasized that there should be realization that the greatest need in radiation therapy today is increased understanding of the fundamental processes by which radiation damages living cells. Possibly the greatest value of new devices will be in the expansion of fundamental studies rather than in direct therapeutic application in radiology.

Jaffe and Zager [31] presented an outline and introduction to some of the uses of radioiosotopes in the diagnosis and treatment of the lesions of the head and neck.

Three groups of cases of cancer, 673 in the tongue, 916 in the buccal cavity, and 550 in the pharynx, treated by radium or x-rays in Manchester, England, were analyzed separately by sex 10 years after treatment. The opinion was expressed by Russell [32] that in the presentation of the results of treatment of malignant disease, failure to state the sex and age distribution may result in fallacious deductions when comparisons are being made between different series of cases. A much better response to treatment is manifest in women than in men. Women are also less likely to have metastasis.

In the treatment of about 500 patients with 2,000,000-volt x-rays, rotational or multiportal methods made it possible, according to Hare and associates,[33] to deliver more adequate doses to the primary lesion and the adjacent lymphatic system. Rotational therapy has been found advantageous for many of the more localized tumors, including those of the larynx, esophagus, and thyroid. The patients tolerated more adequate treatment with less radiation sickness and less cutaneous reaction than would be obtained with conventional x-ray therapy. Hare and his co-workers considered nasopharyngeal tumors unsuitable for rotational therapy.

Lathrop and associates [34] reported their preliminary impressions of the management of malignant lesions of the head and neck, in which the principal method of treatment was radiation with 2,000,000-volt roentgen rays.

In cancer of the paranasal sinuses, radiation produces edema of the mucous membranes and an increase in symptoms with little regression of the tumor, according to MacComb.[35] The orbit should be exenterated, along with curettage of the ethmoid and sphenoid sinuses and resection of the maxilla, if the diagnosis is epidermoid carcinoma. Radiation therapy can be administered postoperatively. Cancer of the posterior third of the tongue or pharynx often can be controlled by irradiation. If activity of the cancer is still present in two to three months, operation should be undertaken.

Cantril [36] discussed the collaborative role of surgery and irradiation in the management of cancer. He stated that irradiation failures are 100% fatal, but radical surgery

offers a possibility of salvaging a significant number of these. In no group of cancers is the close collaboration of surgery and irradiation more important to the over-all prognosis than in many of the more complicated and advanced lesions of the oral cavity.

Freid and associates [37] sounded a note of warning to surgeons concerning the dangers of extensive operative procedures when large volumes of tissue have received high doses of supervoltage radiation. The lack of radiation change in the skin is misleading. Devitalization of tissues is much more marked than after conventional treatment with 200 to 400 kv.

As far as is known today, the use of high-energy radiation in no way alters the fundamental radiobiology of malignant neoplasms. Those tumors which are radioresponsive when radiation of lower voltage is used are responsive to radiation of higher energy. Those which do not respond favorably to radiation with 200 to 400 kv. do not respond favorably to high-energy radiation. Lampe [38] stated that there is evidence to show that high-energy radiation is biologically less effective per unit dose than the lower-energy radiation. The greater depth dose and the "skin-sparing" action can be a deadly combination; there is no skin reaction to function as a warning and the tolerance of mucous membranes and the vasculoconnective tissues of the deeper structures may be surpassed, with serious sequelae.

Nut'all [39] listed several points about the treatment of glands of the neck with radiotherapy: 1. Lesions in these glands vary widely in responsiveness to irradiation, and every treatment is, therefore, somewhat of an experiment. 2. Glands in the middle and upper third of the neck appear to be most amenable to treatment. 3. It is impractical to give to a volume greater than a fourth of the neck a dose sufficient to produce cure. A single mass on one side of the neck is the most profitable lesion to treat. Attempts to implant the whole carotid chain on one side with radium needles or to treat it with roentgen rays is usually disappointing. Implantation of radium needles produces the majority of the few satisfactory results. From time to time, it is suggested that preoperative irradiation will convert inoperable lesions in the glands to operable lesions. That the glands may become smaller and more mobile to palpation is common experience, and technically it may even be possible to excise them. Nevertheless, this operability is an illusion so far as being a cancer-curing procedure.

It is a serious error to permit metastatic lesions in cervical lymph nodes to progress beyond hope of cure while waiting for assurance of success in the therapy of the primary lesion. Control of the metastatic nodes with roentgen rays of conventional voltage has proved unsatisfactory because of the injury to the skin; on the other hand, gamma rays from the radium bomb, the Co^{60} bomb, or supervoltage x-rays will cause palpable nodes to diminish in size or to disappear without significant damage to the skin. Breed [40] indicated that metastatic lesions in the nodes can be controlled by this method until success in the treatment of the primary tumor is assured. Persistently palpable nodes warrant block dissection.

Radiotherapy for metastatic involvement of lymph nodes from carcinoma of the pharynx and larynx should be employed whenever surgical treatment is contraindicated. Antoniazzi and associates [41] pointed out that operations performed a short time after radiation stand a better chance of finding favorable local conditions than could be expected if surgical treatment were effected after a longer period.

Dobbie [42] stated that there is evidence of cervical nodes apparently having been cured to an extent sufficient to justify more reliance than is commonly allowed in the x-ray treatment of nodes unsuitable for block dissection.

A method of treating cervical lymphatic nodes by percutaneous radium implantation is described by Charteris.[43]

According to Paterson,[44] radiotherapy is accepted in Manchester, England, as the primary treatment of intraoral cancer.

Neck

Dissection and Other Surgical Procedures.—The anatomical conglomeration of vital structures in the neck should make the average general surgeon wary of approaching the neck. As Southwick and Slaughter[45] emphasized, those who undertake such operations should be aware of the vital importance of an accurate anatomical knowledge of the field before accepting the responsibility of the treatment of a patient with metastatic disease involving the neck.

Beahrs, Gossel, and Hollinshead[46] describe the technique and present the surgical anatomy behind radical dissection in the neck. This is an excellent paper and should be read by all interested in the anatomy of the neck.

The classic generally accepted anatomical description of the cervical fascia is often presented in so complex and detailed a way that the presentation is of little practical value to the surgeon performing operations in the neck, according to Snitman and Soboroff.[47]

Buxton[48] thoroughly discussed the indications and results of "neck gland resections" as they were performed at the University Hospital, Ann Arbor, Mich., from 1934 through 1954. Five hundred and twenty-two patients had some type of resection of the glands in the neck. It seemed to him likely that supraomohyoid dissection will be used with increased frequency in the future. A more careful and critical selection of patients for bilateral complete removal of nodes must be undertaken. The survival period after bilateral complete resections in the group of patients reported was short and suggests that in many of these the chance of effective cure had long passed at the time the operation was begun.

From a review of the cases of 121 patients subjected to 138 radical dissections of the neck, in 135 operations, Beahrs[49] reported that 74 patients were living and well 3 to 39 months after operation. The operative mortality was 4.4%. The mortality rate can be reduced by performing tracheotomy in combined operations and in those in which dissection is bilateral. Dissection of the neck should be a complete procedure and not be a modified procedure.

Weaver[50] suggested that the term "upper neck dissection" be used for the operative removal of the submental, submaxillary, and superior deep cervical nodes. The term "diagnostic upper neck dissection" indicates that nodal involvement has not been demonstrated clinically.

If Hodgkin's disease is localized to the upper part of the neck in one side, radical unilateral dissection of the nodes of the neck may be justifiable. Craver[51] recommended that this be followed by maximum tolerated irradiation to the entire operative field.

Pressman[52] described a technique for performing radical neck dissection. This technique is based on the premise that the hyoid bone is the surgical keystone of the neck and that removal of one lateral half of this structure, allowing the muscles to remain attached, simplifies and permits an en bloc dissection of all structures between the trapezius and the midline from the mandible to the clavicle to the depth of the third layer of the deep cervical fascia. Hemithyroidectomy also has been added to the routine procedure. This operation is considered justified because of the lymph vessels present in the region of the midline of the neck which are not ordinarily resected.

Gilbert and Feit[53] attempted to simplify and standardize the surgical management of malignant tumors involving the head and neck; they discussed lymph vessels also. To them early diagnosis is the otolaryngologist's responsibility rather than the general surgeon's.

The technique for a complete dissection of the neck and axilla in continuity was presented by Bowden.[54]

Ochsner[55] reviewed the literature and presents the indications for the various

types of dissections in the neck which he performs.

In recent years the one-stage en bloc combined operation on the mouth and neck has been revived. The basis for this procedure is the concept that the so-called ideal cancer operation includes removal of the lymph vessels intervening between the primary focus and the regional metastatic lesion. Such operations are based on the assumption that the mode of lymphatic spread is by permeation. The usual mode of spread, however, is by emboli, and the intervening lymph vessels are free from involvement. Although the combined operation is thus based on a false assumption, Taylor [56] stated that its employment has increased the scope of surgical procedures for, and the operability of, many lesions, notably those of the jaws, floor of the mouth, base of the tongue, and the fauces.

Since metastasis to lymph nodes is primarily due to emboli in the regional nodes, the concept of dissociated therapy is logical under certain circumstances. Control of the primary lesion and later dissection of the lymph nodes, if necessary, are used in squamous-cell epithelioma of the skin, lip, buccal mucosa, tongue, and floor of the mouth, soft and hard palates, and of the maxillary sinuses. In contrast to this method is excision of the primary neoplasm with the regional nodes in continuity. According to Carroll,[57] this is applicable to melanoma of the skin, carcinoma of the salivary glands, thyroid, extrinsic larynx and hypopharynx, and extensive carcinomas of the mouth.

Guiss [58] stated that radical dissection of the cervical nodes in the absence of clinical evidence of metastasis appears indicated for melanoma, papillary carcinoma of the thyroid, and lateralized carcinoma of the tongue.

Ditzler and Eckenhoff [59] found that loss of blood averaged 910 ml. in 29 patients who had radical dissection of the neck utilizing deliberate hypotension and 1415 ml. in 20 control patients. The average operative time was four hours and 45 minutes when controlled hypotension was employed, and three hours and 50 minutes when it was not. Ditzler and Eckenhoff were currently utilizing deliberate hypotension with less enthusiasm than previously.

Pressman [60] stated that it is obvious that bilateral simultaneous radical neck dissection can be performed successfully, but it is equally certain that the weight of opinion is against ligating both jugular veins at the same time.

It is doubtful, indeed, whether bilateral simultaneous block dissection of the neck can ever be justified. Atkins [61] ridiculed the thought: "If surgery be good, bigger surgery is even better." He mentioned a statement attributed to Ogilvie, "If I have a cancer give me a surgeon with plenty of courage, a deal of dexterity and not much sense."

Postoperative Complications.—Gossel and colleagues [62] reported a case in which a chylous fistula developed as a postoperative complication of a left radical dissection of the neck. The fistula was satisfactorily closed with a polyvinyl alcohol sponge.

Gibson and Wollgast [63] report a similar occurrence. Conservative therapy was effective.

Mediastinal emphysema followed by tension pneumothorax is an infrequent but serious complication of operations on the head and neck. Two cases were presented by Verbit and Frelick [64]; one followed thyroidectomy and the other tracheotomy.

Diagnostic Considerations.—Regardless of how careful and experienced the physician may be, his clinical impression of a tumor of the neck is just a guess until he proves the diagnosis by microscopic examination. The attitude, "The tumor is not bothering you, why bother it?" should not prevail, according to Beahrs.[65]

Slaughter and associates [66] stated that (1) exclusive of goiter, most persistent lumps in the neck in the adult are malignant neoplastic disease; (2) most malignant tumors in the neck are metastatic, and (3) most

metastatic malignant tumors in the neck are primary in the head and neck.

Clerf,[67] too, emphasized that any swelling of the neck should be promptly investigated and a diagnosis made.

It is frequently impossible to make a differential diagnosis of a tumor of the neck without the benefit of a biopsy. Baumgartner [68] expressed the belief, however, that certain tumors have characteristics peculiar to themselves by which they may be fairly accurately identified.

Anatomical location plays a large role in the accurate diagnosis of tumors of the neck according to Saltzstein.[69] He presented a series of cases and photographs to show various lesions encountered in the neck.

Hickey [70] presented a general review of masses and tumors in the neck. The role that radical en bloc operations play in the treatment was discussed, too.

Lesions Associated With Congenital Defects.—Lyall and Stahl [71] presented a complete review of the embryology of the branchial system in man. A series of 67 patients with cysts and fistulas of the branchial system was analyzed.

Proctor [72] also completely reviewed this subject. The same subject was discussed by Wilson.[73] The latter is an excellent paper.

Congenital defects of the neck were discussed from their embryonic origin, and the importance of complete excision of the defect to the origin was emphasized by Hunt.[74]

Conway and Jerome [75] reviewed 90 examples of branchial cleft cysts and fistulas. Sixty-two per cent were diagnosed within the first year of life. The recurrence rate following removal of branchial anomalies was 11.1%.

Montreuil [76] presented a brief review of congenital cysts and fistulas.

Comments on five cases of cysts and sinuses of the neck which have originated in or from the first branchial cleft were made by Bill and Vadheim.[77] These cysts and sinuses have extended from just below the middle of the mandible around the angle of the jaw and lateral to the facial nerve, to end at the external auditory canal. Fuller and Murphy [78] reported a similar case of a first branchial cleft cyst presenting in the neck, and Castro [79] reported a case in which the branchial cleft cyst was at the base of the tongue on the left in the pyriform fossa.

Nix [80] analyzed 42 cases of lymphangioma: 10 occurred in the head; 17 in the neck; 1 in the parotid gland and 1 in the submaxillary gland.

Sarason and Roberts [81] reported two unusual cases of cystic hygroma of the neck occurring in the newborn infant with involvement of the tongue and floor of the mouth. Early tracheotomy and operative excision in stages, if necessary, were recommended. Hygroma colli of the newborn infant with involvement of the tongue and floor of the mouth must be recognized as a condition with a high mortality rate. Both Sarason and Roberts' patients died after operative intervention.

Corper and Scanlon [82] reported three cases of congenital cystic hygroma of the neck. They stated that surgical intervention should be undertaken as soon as the infant can tolerate the extensive dissection that is sometimes necessary to excise these tumors.

Jackson [83] presented a brief report of 11 unusual surgical lesions of the neck and a classification of tumors and cysts of the neck.

A case of adenocarcinoma of thyroid tissue originating in a persistent thyroglossal duct was reported by O'Kane and Straus.[84] Two similar cases were found in a review of the literature.

Tumors of the Carotid Body.—The concepts of the biology and treatment of carotid-body tumors were presented by Conley,[85] and Doermann and Meffley [86] made a plea for conservative treatment of such tumors. In their case roentgen therapy proved to be effective.

Birrell [87] reported a case of carotid-body-like tumor arising in the vagus nerve just below the skull in a vagal body tumor. A similar case of a nonchromaffin paraganglioma arising in the cervical portion of the vagus nerve was reported by King.[88] Burman [89] presented a case of vagal-body tumor,

the first, it is thought, to be recorded with widespread secondary dissemination.

Two cases of unilateral tumor of the carotid body were reported by Goormaghtigh and Pattyn.[90] In one case a malignant course extended over nine years. Metastasis was widespread. The incidence of malignancy of carotid-body tumors was estimated at 1.5%.

Romanski[91] reported a case of nonchromaffin paraganglioma of the carotid body with distant metastasis (sites beyond regional lymph nodes). He stated that eight other genuine cases have been reported in the literature.

Turnbull[92] presented a case of malignant carotid body tumor with metastasis to the lungs. The tumor had been considered a neurofibrosarcoma by some pathologists.

A recurrent carotid-body tumor which killed the patient by local extension was reported by Arean.[93]

In a case of bilateral tumors of the carotid bodies, two additional masses were found at necropsy by Marcuse and Chamberlin.[94] All lesions had histological features characteristic of carotid-body tumors. One of the masses was attached to the vagus at a point 5 cm. below the left carotid bifurcation.

Lattes and colleagues[95] presented a case of a large, nonchromaffin paraganglioma of the carotid body of 15 years' duration, in which a second histologically similar tumor developed in the orbit of the same side. It was suggested that this second tumor originated in the "paraganglion ciliare."

Williams, Mason, and Pickard[96] presented a case of carotid-body tumor confused with aneurysm of the carotid vessels.

Three cases illustrating removal of tumor from the carotid body and preservation of the carotid vessels in spite of intimate attachment of tumor to vessels were presented by Farrar and associates.[97]

MacDermott[98] reported a case of carotid-body tumor removed by resecting the bifurcation of the carotid artery.

The results of a consecutive series of 83 carotid ligations were presented by Moore and Baker.[99] The over-all incidence of cerebral complications was 45.5%, and the mortality rate was 30.6%. These authors stated that every carotid ligation depends for its success on an anatomically efficient collateral pathway through the circle of Willis, on the absence of abnormal segmental or diffuse arteriospasm, and on the maintenance of sustained normal arterial pressure and blood volume. The avoidance of hypotension during or following ligation is an important measure. Cerebral complications have been reduced to 31.4% and mortality to 11.4% during the past five years by these authors by paying attention to this factor.

Tumors of Neurogenic Origin.—Tumors of neurogenic origin occurring in the neck are derived from neurectodermal cells of the neurilemma and ganglionic cells of the sympathetic system and parasympathetic systems. The majority of these tumors are benign. There is no specific clinical sign or symptom which will identify these tumors preoperatively. In the surgical extirpation of these neural tumors, every effort should be made to preserve the continuity of the nerve pathway of the involved nerve and associated nerves in that area. Six representative cases illustrating the various types of neurogenous tumors in the neck were described by Conley,[100] to correlate the principles of treatment.

Davis, Edgerton, and Hoffmeister[101] studied a series of cases of neurofibromatosis of the head and neck. They stated that surgical interference does not increase the chances of malignant change. In the head and neck, the tumors of multiple neurofibromatosis are most commonly located in the orbital regions and midlateral portions of the neck.

A case of malignant ganglioneuroma of the nodose ganglion of the vagus nerve was presented by Pack and associates.[102] The anatomical, pathological, and clinical features of this rare entity were discussed.

Wolf and Gilson[103] successfully removed a large bulging neurilemmoma of the neck

from a patient, 77 years old, who had had it for more than 30 years.

Parnes [104] presented a similar case of a neurinoma in the cervical portion of the vagus nerve in a woman 51 years old. The only result of the severance of the vagus nerve was unilateral paralysis of the recurrent laryngeal nerve.

Asherson [105] reviewed the literature regarding solitary neurofibromas of the neck and presented three cases.

Other Lesions.—A case of fibromyxoma developing in the left internal jugular vein was reported by Probstein,[106] who stated that large veins are rarely the site of origin of tumors. Leiomyomas appear to be the commonest type reported. Neurogenic sarcoma and fibrosarcoma have been reported. This is the first instance of fibromyxoma originating in a major vein.

Beck [107] reported a case of myohematoma of the right sternocleidomastoid muscle in a man, 71 years old.

No report of a case could be found which substantiates the opinion that desmoid tumors of the sternocleidomastoid muscle may be malignant. Operation is indicated if after the child is 7 weeks old the lesion continues to grow and is causing deformity, according to Kron and Satinsky.[108]

Oral Cavity and Upper Respiratory Passages

Castigliano [109] observed that, strangely, the oral cavity is a medical "no man's land." The general practitioner in dentistry and the oral surgeons are more concerned with the hard structures of the oral cavity, and the otorhinolaryngologist has been more concerned with diseases of the ear, nose, tonsils, sinuses, the mastoid, and larynx. It must be asserted that a certain minimal standard of diagnostic competence in oral cancer is to be expected from the members of a specialty which should claim the oral cavity as part of its rightful domain.

Usual Malignant Lesions of the Oral Cavity.—The *Cancer Bulletin* (of Texas) [110] estimates that as high as 80% of deaths caused by cancer of the mouth could be prevented by early recognition and prompt adequate treatment. Five-year cure rates by stage of disease are given in Table 1.

TABLE 1.—*Five-Year Cures in Cancer of the Mouth* *

	Early, %	Advanced, %
Gum	39.3	16.7
Palate	38.9	7.4
Tongue	34.8	6.2
Buccal mucosa	35.9	5.7
Floor	29.0	4.8
Tonsil	37.6	7.7
Lips	77.0	29.1

* Based on table in *Cancer Bull.* 7:82-84, 1955.

Slaughter [111] presented the view of a surgeon in a concise manner regarding the treatment of intraoral cancer. To him, obligate indications for surgical treatment of oral cancer are (1) tumors recurring after irradiation; (2) bone invasion; (3) tumors of minor salivary glands; (4) adamantinomas; (5) sarcomas, except lymphosarcoma; (6) radiation cancer, and (7) metastasis to cervical lymph nodes. Preferable indications are (1) superficial lesions; (2) youth, and (3) isolated, keratinizing, squamous-cell lesions of the base of the tongue with pharyngeal involvement. Optional indications are small and superficial oral cancers, particularly of the lateral border of tongue.

Combined resection of jaw and neck was first applied only when there appeared to be nothing to lose, according to Wilde.[112] In light of the results in such cases, indications for use of the operation in more favorable circumstances are constantly being extended.

The present status of intraoral cancer with respect to cause and treatment has been evaluated by Hoover and King.[113] The combined operation should be used more frequently for earlier and borderline disease. Its usefulness in far-advanced disease remains unproved.

Ewing [114] stated in a discussion of the operative treatment of oral cancer that operative treatment will be discredited if it is followed by a high morbidity and by a poor functional result.

The insidious clinical course of tumors of the oral mucous glands may result in complacency of the patient and the consulted

dentist or physician. Smith, Broadbent, and Zavaleta [115] stated that deferment of diagnosis and adequate operation of such lesions will condemn the patient to a life of inconvenience, if not to eventual death from tumor. They presented 32 cases.

Castigliano and Rominger [116] studied 752 cases of oral cancer. Seventeen patients died from visceral metastasis, an incidence of 5.3%. With an increasing ability to control the local lesion together with the metastatic lesion in the cervical nodes, they have noted an increase in the life span after treatment in fatal cases. Coincident with an increased survival time in these patients, distant metastasis is being observed with greater frequency.

Four hundred and one cases of intraoral squamous-cell carcinoma were analyzed by Tiecke and Bernier.[117] Only 8% of the patients with carcinoma of Grade 4 (Broders' classification) survived two years. Fifty-two per cent of all the carcinomas involved the tongue. Of 372 patients, 21 survived five years or more.

Kutscher and associates [118] found a chlorophyll-containing solution to be ineffective as a deodorizing agent in cases of advanced or terminal carcinoma of the oral cavity.

Ward and Chambers,[119] Briggs [120] and Byars [121] presented their views in separate papers on the management of intra-oral cancer and its treatment with radical surgery.

Shedd [122] emphasized the importance of handling patients with intraoral cancer as a team enterprise, combining the specialized knowledge from several branches of medicine.

While the treatment of malignant lesions is primarily the problem of the medical profession, those of the oral cavity are seen more frequently by the dentist. A dentist should be included in clinics organized for the detection of cancer according to Salman and Langel.[123]

The *Cancer Bulletin* (of Texas) [124] stated that in few other diseases does even the slightest delay so affect the ultimate prognosis as in cancer of the tongue.

Survival Rates and Treatment of Carcinoma of the Oral Cavity.—Intraoral and pharyngeal neoplasms together account for 45% of all malignant growths seen in Indian patients at the Tota Memorial Hospital, according to Paymaster.[125] Cancer of the tongue was the most frequent intraoral cancer; next in frequency was cancer of the buccal mucosa. Six hundred and fifty patients were studied in detail. Sixty-eight per cent had metastastic lesions in the neck. Radiation and surgery produced 54% five-year cures.

Marchetta and Mattick [126] presented the results of irradiation as almost the only treatment used in 247 cases of carcinoma of the tongue. No radical surgery was done. The absolute five-year survival rate was as follows: oral portion, 19.2%; without involvement of the nodes of the neck, 29.3%; and base of tongue, 9.8%. The more differentiated lesions gave a higher survival rate than the less differentiated ones. Irradiation cured only an occasional patient with metastatic lesions in the neck.

Kremen [127] reported on a series of 23 patients operated on for cancer of the tongue at the University of Minnesota. He advocated en masse removal of the primary lesions, intervening tissue, and regional lymph-bearing regions. Of 10 patients so treated more than five years before his writing, 70% were alive and free of disease. Of five patients with pathologically involved nodes, two were alive and well five years later. Of five patients with pathologically negative nodes, five were alive and well five years later.

Rahausen and Sayago [128] analyzed 113 cases of cancer of the tongue treated at the Radium Institute, Santiago, Chile. Metastasis to lymph nodes developed in 75.2% of all cases. Lesions of the anterior two-thirds of the tongue were treated with interstitially applied radium needles and those of the posterior one third with external roentgen irradiation. Prophylactic dissection of the neck was advised. The five-year cure rate was 8.1%.

In a study of 94 cases of cancer of the tongue, Martin [129] found an over-all five-year cure rate of 32% obtained with a non-surgical method based on the treatment of the tongue lesion with implanted radium needles of low intensity and the use of the same needles and x-ray therapy for metastatic lesions in the cervical nodes.

In a separate paper Martin and Martin [130] stated that in a group of 40 cases of cancer of the posterior third of the tongue treated primarily by irradiation, a five-year salvage of 38% was obtained. The primary lesions were implanted with radium needles of low intensity while the involved cervical nodes were managed by a combination of radium-needle and x-ray therapy. No block dissections were done.

Krantz, Grady and Holloway [131] analyzed the results of treatment of the cases of cancer of the tongue and floor of the mouth at the Veterans' Administration Hospial, Atlanta, Ga. More than a third of the 83 patients were apparently free from disease from 2 to 12 years after treatment. The authors stated that treatment must be individualized, and that certain cases lend themselves to radical block excision of all disease as the initial treatment. The various types of lesions were classified and the form of treatment used for each type was described.

To Ash and Millar [132] the best initial treatment of the primary lesion in cancer of the tongue and floor of the mouth has seemed to be radiotherapy. Better results appeared to attend use of radium or interstitial irradiation with radium plus roentgen rays than external therapy alone. Gross, five-year survival rate for 95 patients with cancer of the floor of the mouth was 38.9%. For 394 patients with cancer of the tongue it was 27.2%; in 44.3% of the patients with cancer of the floor of the mouth and in 55.0% of the patients with cancer of the tongue, lesions developed in the cervical nodes.

In the light of experience gained from a series of 131 cases of carcinoma of the floor of the mouth, in all but three of which radiotherapy was employed, Windeyer [133] has begun to submit more patients to radical surgical excision. Cases of carcinoma of the alveolus with erosion of bone are more suitably treated in this way.

Gardham [134] stated that the over-all cure rate for cancer of the floor of the mouth recorded after various forms of treatment is constant in the neighborhood of 28%. The rarity of good palliative results was stressed and it was suggested that patients whose lesions appear to be unfavorable as a result of local extension are more suitable for operation than irradiation.

Dobbie [135] was of the opinion that the evidence as a whole shows a cure rate from indicated treatment of 60% in early cases of cancer of the floor of the mouth and in cases in which lesions do not develop in the nodes, and of 30% in cases in which block dissection is indicated and also in the whole group of cases.

Early and small localized cancers of the floor of the mouth should be treated by radiation only, according to Cade.[136] Local involvement of tongue and gums requires locally applied diathermy in addition. For involvement of bone and widespread disease in the mouth, surgical excision offers a better chance. Operation should be on a large scale. Methods using tube pedicles are badly tolerated by patients with cancer of the mouth, and other methods, quicker in achievement, although of inferior cosmetic effect, should be employed.

Rahausen and Sayago [137] studied 40 patients with malignant tumors of the floor of the mouth treated at the Radium Institute of Santiago, Chile, between 1930 and 1949. Of the entire group, 27.5% had metastatic lesions. Roentgen therapy applied externally produced no successful results. It was their opinion that intraoral surgical procedures have no place in the treatment of cancer of the floor of the mouth. They stated that if surgical treatment is to be used, it should be en bloc resection of the tumor plus the adjacent portion of the tongue and half the mandible. They performed prophylactic suprahyoid dissection of the neck because

of the difficulty of follow-up. Their best results were obtained by volume implantation of radium needles followed by external radium. Five-year cure rate in 38 cases was 15.7%.

In those parts of India where the habit of chewing tobacco and keeping tobacco with lime and areca nuts in the groove of one cheek is rampant, the incidence of carcinoma of the cheek is higher than the incidence of other oral lesions, according to Srivastava.[138] The largest number of cases come from Mainpuri where the tobacco, lime, and areca nuts are kept on one side of the mouth for hours even during sleep. Tobacco is thought to be harmless unless mixed with lime. The chemical irritation is due to alkaloids produced by the action of lime on tobacco chewed with areca nuts and betel leaves.

Handy and Goldberg[139] investigated the occurrence of malignancy in children. From 1949 to 1951, nine children less than 15 years of age were reported to have had malignant tumors of the buccal cavity in New York State exclusive of New York City, and 6 out of a total of 599 had malignant tumors of the respiratory system. The commonest tumors were leukemias (35.7%).

A case of epidermoid cancer of the tongue and floor of the mouth in a 4-year-old child was reported by Merrifield, Dalitsch and Steiner.[140]

Fifty previously untreated carcinomas of the buccal mucosa in all stages of the disease were irradiated by Lampe.[141] Of 30 eligible, 29.6% survived five years. Of 14 patients showing unequivocal destruction of bone, 28.6% were without recurrence at five years.

Breed[142] presented the results of the treatment of cancer of the buccal mucosa with radium and radon in 42 cases at the Breed Radium Institute. The five-year cure rate was 35.7% between the years 1930 and 1949.

Modlin and Johnson[143] reported the results of surgical treatment of epidermoid cancer of the buccal mucosa and lower gingiva of 63 previously untreated patients. Thirty-six of these patients were treated by jaw-neck dissection. A five-year absolute control rate of 47% was obtained after 23 jaw-neck dissections for carcinoma of the buccal mucosa and lower gingiva.

Ulin and associates[144] reported a case of rhabdomyosarcoma of the tongue and presented a comprehensive review of the pertinent literature. Their patient was alive and well four years after radical excision of the tumor.

Gilbert[145] reported a case of cylindroma of the tongue.

Bogdasarian[146] recommended the extraction of teeth in the pathway of radiation prior to x-ray treatment if this means of treatment is to be used for oral cancer. If the fields are to be large, all teeth should be extracted.

Benign Lesions of the Oral Cavity.—Dixon and Ziskind[147] reported a case of lipoma of the buccal mucosa.

Pollack[148] reported a case of median rhomboid glossitis in a 9-month-old white infant, and Sirsat[149] reported six cases. It is generally accepted that no treatment is indicated. This lesion is frequently confused with carcinoma.

Fibroma of the frenum under the tongue, or Riga's disease, is rarely mentioned in the literature. It is a neoplasm which may extend to 2 cm. in diameter. It disappears spontaneously after a few years. Shah[150] reported a case in a child, one year old.

Burston[151] presented a case of recurrent swelling of the tongue due to cirsoid aneurysm.

Two cases of chondroma of the tongue were reported by Bruce and McDonald.[152]

Fatin[153] reported a case of unilateral macroglossia due to neurofibroma, treated by hemiglossectomy. A similar case removed by an external transverse cervical incision was reported by Harpman and Prior.[154]

Plexiform neuroma of the tongue may cause unilateral enlargement of the tongue. The condition is usually present at birth

or in infancy. Bird and Clark [155] reported two cases and reviewed the literature.

The oral manifestations of von Recklinghausen's disease are changes in the mucous membrane, associated lesions of the facial bone, and dental changes. Macroglossia is common, according to Baden and associates.[156] Single nodular tumors may be present. Changes in the maxilla and mandible are rare. These may be asymmetry or hypertrophy, radiolucent defects, or atrophic changes.

Macroglossia associated with primary systemic amyloidosis was reported by Caudell and Yoshida.[157] A successful one-stage palliative partial glossectomy was performed.

Hendrick [158] performed partial glossectomy for macroglossia produced by congenital lymphangioma.

Salman and Langel [159] reported cases of benign osteogenic tumors of the oral cavity.

Certain oral lesions are associated with Hand-Schüller-Christian disease. Stahl and Robertson [160] presented a case and reviewed the literature.

Malignant Melanoma.—Malignant melanoma of the oral cavity is rare. Moore and Martin [161] summarized the total experience with melanoma of the upper part of the respiratory tract and oral cavity at Memorial Hospital, New York, between 1930 and 1948. Twenty-six cases were available for study. The net five-year cure rate was 4.5%. If the arbitrary limit of five years were extended to six years, none of the 26 patients could be considered cured of disease. The importance of early recognition of melanoma by the physician is emphasized.

Catlin [162] stated that the surgical policy at the same hospital is to excise adequately the primary melanoma without sacrificing an excessive amount of adjacent tissue. A block dissection of the regional lymphatics is made only when metastatic lesions are present or suspected or when excision of the melanoma requires surgical entry into the area of lymphatic drainage; for example, excision of a melanoma in the skin of the neck would be extended to include dissection in the neck as an integral part of the operation. No effort is made to perform the local excision and neck dissection in continuity except in instances in which such an operation would be technically convenient. Radical dissections in continuity are anatomically impractical for many melanomas of the head and neck; he expressed the belief that this principle has been overemphasized and is actually of trivial importance in successful treatment. The five-year cure rate for melanomas of the skin of the head and neck is 36%. The prognosis depends on the absence or presence of metastatic involvement at the time of initial treatment.

Stewart, Hay, and Varco [163] studied 92 cases of melanoma treated at the University of Minnesota Hospitals since January, 1932. In 35 (38%) of the 92 cases, lesions occurred in the "head and neck"; in 14 of 35 cases, on the cheek.

Alexander [164] reported a case of malignant melanoma of the nasal septum. Radical surgical excision should be practiced at the earliest possible moment if any type of ultimate survival is to be obtained.

Thirteen malignant melanomas of the nose and ear were studied by Mason and Friedmann.[165] All were resistant to both surgical and radiological treatment. Pigmentation was present in all cases, though in varying amounts.

Baldridge and Waldron [166] reported a case of melanoma of the left upper jaw and hard palate.

Plasma-Cell Tumors of Upper Part of Respiratory Tract.—The majority of extramedullary-cell tumors reported have been found to have their origin in the upper part of the respiratory tract or oral cavity. Caution must be exercised when dealing with these tumors. Carson, Ackerman and Maltby [167] stated that their course is unpredictable and that they may remain stationary for many years before they develop

into diffuse myeloma or expand rapidly as an invading type of tumor, destroying bone and other tissues of the affected area.

Priest [168] presented a case of multiple benign plasmacytoma of the upper air passages. As long as the plasma-cell tumor is localized and confined to soft tissue, a clinical cure may be obtained by surgical removal or irradiation or both. On the other hand, therapeutic measures rarely have prevented a fatal outcome if applied after the tumor has either locally invaded bony structures or spread more distantly to lymph nodes or to the skeleton.

Schwartz [169] stated that plasmacytoma is a cancerous disease. A latent phase may last from a few months to many years, but eventually generalized disease (multiple myeloma) ensues. The average period of survival after the process has become generalized is the same (two years or less) as that in multiple myeloma, with exceptional prolonged remissions in both. The location of the primary growth is a determinant in the duration of the latent period. Primary lesions in or adjacent to bone have, on the average, a short latent period, and the survival expectancy is poor. Histological appearance gives no indication of the future clinical course of the disease. The use of the term "benign plasmacytoma" is unjustified except in plasmacytic granuloma. Radiosensitivity is not a universal characteristic of the tumor. Many are, however, exceedingly sensitive to roentgen therapy and radium. Radiosensitivity of the primary tumor is a favorable prognostic sign.

Heatly,[170] too, indicated that the vast majority of primary plasma-cell tumors in the upper air passages are variants of multiple myelomatosis. The radiosensitivity of these tumors favors radiotherapy. Experience does not appear to justify the radical surgical procedures commonly employed in the treatment of the more familiar malignant neoplasms encountered in this region.

Howarth [171] reviewed the pertinent literature and presented five cases of plasmacytoma of the upper part of the respiratory tract. The primary site was in the nasopharynx in two instances, in the nasal cavity in one, in the maxillary sinus in one, and in the pharynx and larynx in one as multiple polyps.

Two cases of myelomatosis were described by Emslie-Smith and associates.[172] In each, an extramedullary plasmacytoma presented in the upper air passages, and later numerous widespread cutaneous deposits developed.

Taylor and associates,[173] Korkis,[174] and Jay,[175] in separate papers, presented case reports of plasmacytoma of the upper air passages.

Paranasal Sinuses

Malignant Disease.—Despite the encouragement due to improvements in surgical technique, cancer of the paranasal sinuses remains a highly lethal disease, and its treatment is one of the most difficult of all lesions encountered in the region of the head and neck. The continued high mortality rate can be attributed mostly to the complex anatomy of the nasal cavity and accessory sinuses, which makes early diagnosis of these neoplasms almost impossible. The results of treatment, regardless of the method, are often poor and usually mutilating. Recent statistics compiled on the Head and Neck Service of the Memorial Hospital by Frazell [176] indicated a five-year cure rate of 22.5% based on 173 determinate cases treated during the years 1935 through 1947. These patients were mostly treated by a combination of surgery and irradiation. It is their belief that more emphasis should be placed on surgical procedures and that irradiation should be reserved for postoperative use when the lesion is advanced and unfavorable or when there is clinical evidence of residual or recurrent disease.

Larsson and Martensson's [177] study of carcinoma of the paranasal sinuses should be read in toto by students of the disease.

The investigation relates to 379 cases of carcinoma of the paranasal sinuses and nasal cavities treated from 1940 to 1950 at Radiumhemmet and the Otolaryngologic Department, Karolinska Sjukhuset, Stockholm. The following five-year results were obtained: The absolute five-year cure rate was 23%; the cure rate of all surgically treated patients (electrocoagulation in combination with radiotherapy) was 45%; that of patients subjected to radiological treatment alone (largely inoperable lesions) was 12%.

They gave the principles of treatment at Radiumhemmet as follows: Every patient is evaluated jointly by an otologist and a radiologist. Each patient has a biopsy. Patients whose lesions are considered operable receive preoperative roentgen irradiation and are submitted to radical operation four to six weeks later by electrocoagulation. Radium tubes are inserted at the same time in the operative cavity.

Mattick and Streuter [178] surveyed the records of the Roswell Park Memorial Institute covering epithelial malignant lesions of primary origin arising in the maxillary antrum. In the period 1939 through 1946, 68 cases of cancer of the antrum were noted. The future trend of treatment of these tumors at this institution seems to be radical operation or electrocoagulation supplemented by irradiation or both. Treatment in this series was mainly by radiation, often combined with conservative surgery. The absolute five-year cure rate was 15% in all cases, with a relative rate of 35% in early cases.

Wilson [179] favored preoperative irradiation followed by electrosurgical removal of the palate and insertion of radium whenever necessary. The results of treatment still leave much to be desired. He has found that in more than a third of the cases no growth is discernible histologically in the material removed at operation after irradiation.

Experiences with 122 cases of cancer of the paranasal sinuses and nose at three different hospitals were reported by Turnbull and Hara.[180] Eighty per cent of the lesions arose in the maxillary antrum. They gave two logical reasons for surgical treatment before irradiation: (1) there is less tumor mass to irradiate, and (2) drainage has been established for the products of tumor necrosis, irradiation, and infection.

Cranmer [181] presented a preliminary report of 91 cases of malignant neoplasms of the paranasal sinuses treated at the University Hospital, Ann Arbor, Mich., from 1932 to 1948. A 14% five-year survival rate was obtained. Emphasis in this paper was on diagnosis because much valuable time is lost before definitive treatment is begun.

Data concerning 16 patients seen at the Ohio State University Medical Center with primary carcinoma of the maxillary antrum were presented by Raines and James.[182] They advocated adequate surgical resection, the application of a plastic mold containing radioactive substances to the defect to provide the necessary irradiation.

Hendrick [183] recommended preoperative irradiation followed by radical resection of the maxilla with or without exenteration of the orbit for cancer of the nasal cavity and paranasal sinuses.

Weaver [184] stated that little more can be done to facilitate the earlier diagnosis of malignant tumors of the maxillary antrum with our present methods. The use of surgical diathermy is recommended for the treatment of malignant tumors of the maxillary antrum.

Smith and associates [185] described a surgical procedure that allows en bloc removal of frontal ethmoid cancer. The problems involved in cancer of a paranasal sinus were described.

Priest and Kucera [186] described maxillectomy through the Fergusson incision when frozen sections are used as a guide to the extent of removal necessary. The history of the Fergusson incision was reviewed also.

Poulsen [187] advocated electrocoagulation and radium tubes followed by x-ray therapy for malignant tumors of the nose and paranasal sinuses. In the future, he intended to use x-ray irradiation preoperatively, too. He presented a series of 19 patients operated on, of whom 12 were dead and 7 were still alive. Only five patients were observed more than five years after treatment.

Winborn [188] reviewed the literature and analyzed some typical cases of malignant diseases of the nose and sinuses. The methods most satisfactory in the treatment of 26 cases of malignant disease were outlined. Best results seem to have been obtained by combined electrosurgery, intracavitary radium, and external irradiation.

No radiological features seem peculiar to malignant tumors in the region of the nose. The radiological findings and the technique employed in the examination of malignant tumors of the nose, accessory sinuses, and nasopharynx were described by Gordon.[189] The contribution of radiology to the diagnosis and management in such cases was discussed.

Mårtensson [190] stated that carcinoma arising from the mucous membrane of the maxillary sinus is not always associated with roentgenographically demonstrable destruction of bone. Large maxillary cysts may be attended by bony destruction typical of carcinoma.

Biopsy is an essential aid to diagnosis of intranasal lesions, but Seltzer [191] emphasized that, if the result of a single biopsy and the clinical data do not correspond, repeated tissue examinations are in order.

Bear and Kowal [192] described a simple inexpensive temporary prosthesis to be used after maxillary resection. It is composed of a polyvinyl plastic sponge attached to an acrylic or plastic base or to the patient's own dentures.

Skin grafting the postoperative cavity after resection of the maxilla for malignant disease is a recognized procedure. Lynch [193] did not favor this because a graft would cover the area and might conceal evidence of early recurrence.

Conley [194] presented the rationale and technique of free skin grafting to the sinus cavity, the internal aspect of the cheek, alveolus, buccoalveolar and glossoalveolar sulci, the tongue and floor of the mouth, the palate, pharynx, larynx, and cervical esophagus.

Radical surgery plays an important role in the treatment of extensive and recurrent malignant lesions of the face and scalp. Masters and associates [195] emphasized that the acceptance of postoperative deformity is essential in the treatment of such lesions.

In a separate paper by Maguire and associates [196] on various lesions of the cheek, the etiology, differential diagnosis and various methods of treatment were presented. Locations and types of incisions were pictured, and a new suborbital approach was described.

Rainer and Burne,[197] and Sciarra and Hallberg [198] in separate papers reported cases of carcinoma of the frontal sinus.

Ratko [199] presented his opinion regarding the diagnosis and treatment of tumors of the nose and paranasal sinuses.

Marks [200] reported two cases of advanced malignant lesions of the antrum treated by roentgen-grid therapy in one instance and by telecurietherapy in another. One patient has survived five years (epidermoid carcinoma) and the other has survived 20 years (spindle-cell sarcoma).

Takashi [201] presented the histogenesis and classification of carcinomas of the paranasal sinuses.

McMahon [202] reported a case of squamous-cell carcinoma of the maxillary sinus in a man aged 20, and Hibbs and Teloh [203] a case of mucus-producing adenocarcinoma of the ethmoid sinus.

Three uncommon tumors were reported by Takashi [204]: a primary adenocarcinoma presumably arising from the ethmoid sinus typified by rosettelike glandular formation; a malignant melanoma of the ethmoid sinus, and ganglioneuroma from the nasopharynx.

A case that illustrates the value of persevering with curative treatment in spite of fairly heavy odds against an ultimate cure was reported by Stratton.[205] For two years, the patient was treated for sarcoma of the ethmoid sinus which recurred twice and for brain abscess which recurred once; however, he has now been alive and free of disease for three and one-half years.

Soboroff and Lederer[206] reported a case of chondrosarcoma of the nasal cavity. The diagnosis and classification of tumors of cartilaginous structure often present many difficulties.

Miscellaneous Conditions.—Struthers, Williams and Parkhill[207] reported a case of papillary cystadenoma (Warthin tumor) of the maxillary paranasal sinus. To their knowledge, the finding of a Warthin tumor arising in the maxillary paranasal sinus has not heretofore been reported.

Peimer's[208] brief review of the literature on benign giant-cell tumors demonstrated the paucity of cases of benign giant-cell tumors that involve the nasal sinuses and skull. In the three cases presented, such tumors involved the maxillary sinus, the sphenoid and ethmoid bones, and the roof of the orbit. Complete excision seems to be the best treatment.

Keller and colleagues[209] reported a case of giant-cell tumor of the maxilla. To them preoperative x-ray therapy might be of some benefit in reducing hemorrhage during the surgical removal of extensive giant-cell tumors.

Livingstone[210] stated that sarcoidosis is a systemic disease due to infection and that the nasal mucosa, especially over the inferior turbinates, is commonly affected and may even be the portal of entry. A case of sarcoidosis of the maxillary antrum was presented.

A case of acute myeloid leukemia presenting as acute frontal sinusitis was presented by Dawes and Levene[211] merely as a reminder that acute leukemia may present as an infective disease.

Mucocele.—Simon and Tingwald[212] stated that a mucocele of the sphenoid sinus produces a definite syndrome with associated radiographic evidence sufficient for clinical diagnosis. Rhinologic examination usually is negative, although some edema of the turbinates or other signs of sinusitis may be found and slight bulging of the anterior wall of the sphenoid sinus may be observed. Two cases were reported with radiographic findings.

Routine roentgen examination of a frontal sinus that has been subjected to radical operation will disclose only operative and postoperative changes. After injection of contrast medium into the nasal cavity on the affected side the roentgenogram will show whether or not the newly constructed nasofrontal passage is open. If a mucocele is present, the picture varies according to whether the contrast medium is injected into or outside of the mucocele. Barr, Koch, and Welin[213] presented a new technique for injecting medium through the old trephine opening.

Tamari and Bear[214,215] reviewed the literature on mucocele of the frontal sinus, reported on 20 cases, and discussed the various surgical techniques employed in the treatment of this uncommon lesion in this site and in other sinuses. They recommended radical external frontal approach for mucocele of the frontal sinus.

Osteoma, Fibroma, and Cysts of Frontal Bone.—Murphey[216] submitted evidence that osteoma of the frontal sinus is not rare.

Shaw[217] reported a case of ethmoid osteoma. The clinical features leading to the detection of the tumor are unusual in that they resulted from the direct introduction of air into the cranial cavity through dural discontinuity. The intracerebral pneumatocele so formed was directly due to erosion of the dura mater in the region of the right cribriform plate by a large osteoma which was placed high in the posterior ethmoid region. There were no symptoms directly referable to the nose or sinuses.

Andrew [218] said that early excision of osteomas is desirable, and in many cases this should be through a frontal craniotomy.

The part played by interference with the drainage of the frontonasal duct in the causation of symptoms in cases of osteoma was emphasized by Howells and Jones.[219]

Rowbotham, Clarke, and Hammersley [220] described and illustrated a lateral surgical approach for removal of a large bony osteoma of the frontal sinus that had eroded the orbit to cause cellulitis and proptosis.

Skolnik and Fornatto [221] reported four cases of ossifying fibroma involving the paranasal sinuses.

The frontal bone can be the seat of cavities other than the frontal sinuses. These cavities or cysts are produced by different diseases, the nature of some of which may be difficult to ascertain. Handousa [222] reported four such cysts.

Caparosa [223] reported a case of epidermoid cyst of the frontal bone with communication to the frontal sinus. Craniectomy and radical excision were necessary to produce cure.

Nose and Nasopharynx

Stevens [224] recorded his experience with all cases of malignant tumor of the nasopharynx treated at the Cauliff Royal Infirmary from January, 1948, to August, 1954. Thirty-five cases were available for study. In this series exploration through a transpalatal incision was done in five cases. Although excision of the primary lesion was not usually possible, except perhaps for some mixed salivary growths, this approach was useful in finding the extent of the lesion and in providing a permanent palatal window for more efficient irradiation. In seven cases of this series, block dissection of the neck was carried out.

Mattick and Marchetta [225] presented the results of the treatment of 82 patients with cancer of the nasopharynx: 18.9% were free of disease five years or more after initial therapy; 31% of the patients with lymphoepithelioma survived five years or more; 17% of those with epidermoid type of lesion survived five years or more; 33% of the patients without involved cervical nodes survived five years or more, whereas 18.9% with involved nodes survived that period. They recommended intracavitary irradiation of 200 mg. hours repeated every other day for three periods followed by 6000 to 7000 r through two fields.

Although irradiation is the treatment of choice for both sarcoma and carcinoma of the nasopharynx, surgical extirpation may be indicated in other types of growth, according to Hara.[226] He described transpalatine resection of nasopharyngeal growths.

McWhirter,[227] Gooddy,[228] and Cawthorne [229] in a symposium emphasized the importance of early diagnosis in cancer of the nasopharynx.

Cranial nerves were involved in 48% of 185 cases of nasopharyngeal carcinoma seen over an eight-year period in Singapore. The abducens and trigeminal nerves were involved most frequently, according to Lawley.[230]

Mekie and Lawley [231] presented a clinical analysis of 120 cases of nasopharyngeal carcinomas. Das and associates [232] and Duboe [233] in separate papers presented data on a series of 16 and 4 cases respectively.

Van Herik and Erich [234] described an intracavity nasopharyngeal radium applicator, which is a useful supplement to x-ray treatment of squamous-cell carcinoma of the nasopharynx.

Weaver [235] reviewed the literature for the past 25 years as it applied to lymphoblastoma and to the otolaryngologist. Brief case reports are made to illustrate the variety of cases encountered.

Towson [236] presented a case of reticulum-cell lymphosarcoma of the nasopharynx, and Shuster and Shuster [237] presented another arising in the paranasal sinuses.

The choristoma represents displacement of tissue in the course of development so that it appears in a neighboring organ.

Gundrum and co-authors [238] reported a case of choristoma of the nasopharynx.

Schindler, Hurwitz, and Greenhood [239] reported finding a teratoid tumor in the nasopharynx of the newborn.

Rao [240] recorded a nasopharyngeal chondroma that filled the nasopharynx, which was primary in the right Eustachian tube of an 18-year-old youth. The tumor was removed by the transpalatal approach.

The origin of nasopharyngeal chordoma was described briefly by Crikelair and McDonald.[241] A case in which there was marked destruction and distortion of the face, the results of palliative operation and irradiation therapy, was presented.

Radcliffe [242] described an unusual case of transitional-cell papilloma occurring in the nasal and oral portions of the posterior pharyngeal wall. It was removed by diathermy excision through a transpalatal approach.

Rowland [243] and Batory [244] in separate papers reported single cases of lymphoepithelioma of the nasopharynx.

Epstein [245] described a case of nasopharyngeal fibrosarcoma in a 3-year-old boy who had no symptoms referable to the nose. The clinical picture was that of a posterior fossa tumor.

St. John and Woo [246] presented a case of rhabdomyosarcoma of the nasopharynx in a 4-year-old child.

Burns and associates [247] reported a case of renal carcinoma metastatic to the nasopharynx, and Achar [248] reported a case in which nasal metastasis of hypernephroma caused recurrent episodes of epistaxis.

Taylor and Burwell [249] reported two cases of bilateral and two of unilateral nasopharyngeal cysts. The question of bronchiogenic origin was discussed, and previous publications were reviewed.

Mills [250] described a midline cyst of the nasopharynx removed by a transpalatal approach.

Hoch [251] presented a patient with cystic craniopharyngioma who had been treated by two transfrontal craniotomies without preventing enlargement of the the tumor. The endonasal approach is considered to have definite application in the treatment of cystic craniopharyngiomas.

Handousa, Farid and Elwi [252] discussed some of the controversial aspects of nasopharyngeal fibroma, basing their views on 70 authentic cases encountered in the last 20 years. They are of the opinion that the lesion is a true tumor and not an allergic condition or inflammatory lesion. Their cases prove that the disease is not exclusive to males or to the age of puberty, provide good evidence against the theory of uniform regression at sexual maturity, and favor the view that the tumor originates from the periosteum. Surgical excision was considered the ideal method of cure. They recommended lateral rhinotomy, but did not favor irradiation in any form or surgical diathermy. They have operated on 61 patients with an operative mortality of 3.3%.

Sternberg [253] made a pathological study of 25 cases of juvenile nasopharyngeal angiofibroma. He said that these tumors are sex-linked and age-linked and probably are influenced by some as yet unknown endocrine factors.

Hemley, Schwinger, and Friedman [254] stated that radiation therapy may be a life-saving procedure in a patient with severe bleeding from a nasopharyngeal fibroma or if the general condition of the patient is poor. Radiation therapy may be employed prior to operation in an attempt to decrease the vascularity of the tumor. They presented one case in detail and mentioned two others in which treatment with x-rays was used.

Erich [255] advocated treating nasopharyngeal fibromas with radon seeds.

Kremen [256] presented two cases of nasopharyngeal fibroma. A technique is presented for surgical managment of angiofibromas of the nasopharynx by lateral nasopharyngostomy which entails the transection of the ascending ramus of the mandible.

Martin[257] described a transpalatal approach to the nasopharynx with permanent fenestration of the palate if necessary for the treatment of nasopharyngeal fibroma.

White[258] advocated a combined intranasal and postpalatal approach.

Dane[259] presented a case of juvenile fibroma of the nasopharynx in a man of 31 years. The pathological and clinical features of the tumor, said to be in a state of regression, were emphasized.

Batsakis, Klopp, and Newman[260] presented a case of supposed "juvenile" nasopharyngeal angiofibroma in a 54-year-old man for which partial surgical removal, extensive irradiation, and subsequent surgical excision were employed. Areas of fibrosarcoma were found in this tumor. The nasopharynx was approached by splitting the mandible and lower lip in the midline and continuing the incision up the gingivolingual sulcus, elevating the mandible and cheek laterally, thus exposing the pterygomaxillary area and the nasopharynx.

Simple fibromas of the nose are uncommon tumors. The majority of fibromas of the nasopharynx described have been of the juvenile type. In Farrar's[261] unusual case, a simple fibroma arising from the middle turbinate of the nose presented in the nasopharynx; the patient was a boy aged 13 years.

Intranasal encephalomeningoceles are usually considered congenital herniations of the brain and meninges. The diagnosis should be established before any operative procedure or biopsy is done. Moore[262] stated that this can be accomplished by observing the following points: (1) abnormal width between the eyes; (2) cystic polypoid mass in the nose with attachment between the middle turbinate and septum or to the septum and anterior walls of the nasal cavity, and (3) mass present from birth. He also said that roentgen evidence of bony cranial defect is seldom seen, that brain pulsations and increase in size on crying are not seen, and that intracranial repair of the bony dehiscence combined with intranasal removal of the herniated mass is advocated.

Figi, Craig, and Kurth[263] presented a case of intranasal encephalocele in a girl 15 years of age. The encephalocele was successfully removed via an extracranial approach which involved ligation and removal of the sac and lining the cavity with a skin graft. An external incision through the bridge of the nose was used.

Cobb[264] also reported a case of nasal encephalocele. He stated that encephalocele should be considered when a cystic lesion of the nose is seen in early childhood. Abnormal width of the nose or forehead and the finding of sugar in fluid draining from the nose similar to that of spinal fluid support the diagnosis.

Cases of frontonasal glioma are reported by del Villar,[265] Cassidy and Wahl,[266] Dawson and Muir,[267] MacDonald,[268] Day,[269] Low and co-workers,[270] and MacNeil.[271] Such tumors are of congenital nature and embryonal origin. They behave like benign tumors, and there is no reason that they should recur, except perhaps as a result of incomplete surgical removal.

Tumors of the nasal fossa apparently related in origin to the olfactory area have been neglected by American pathologists, although several reports of their occurrence can be found in the European literature. Five cases were presented by McCormack and Harris.[272] These were called "neurogenic tumors." They arose in the lateral nasal wall. Malignant courses terminated in the death of two patients in less than two years. One tumor was classified as an olfactory neuroepithelioma, as true rosettes were formed, and the other as an olfactory neuroblastoma because of the presence of pseudorosettes. In the other three cases the clinical course has been benign since adequate surgical removal of the tumors. A uniform cellular pattern and abundant production of fibrils characterized them histologically. Neoplasms in this group were designated by the authors as "olfactory neurocytomas."

Frühling and Wild [273] presented four cases of true olfactory esthesioneuroepitheliomas of the nasal fossae. Neurogenic tumors of the nasal fossae originating in the olfactory organ are infrequent, but it is probable that their rarity is partially due to failure to recognize them (owing to the possibility of histological confusion with glandular epitheliomas or sarcomas). These authors stressed the "attenuated" malignancy of these tumors, their extreme radiosensitivity, and the considerable length of survival of the carefully treated patient.

Fisher [274] reported a case of neuroblastoma of the nasal fossa. The patient died 10 years after onset of symptoms and primary excision of the nasal tumor. The literature on these lesions was reviewed. These tumors are capable of widespread metastasis and warrant guarded prognosis. Their radiosensitivity was reemphasized.

Granuloma.—Hanbery and Rayport [275] summarized the clinical and pathological features of nine cases of orbitofrontal cholesterol granuloma. All lesions were solitary and occurred in young or middle-aged males. Unilateral exophthalmos and diplopia were the commonest symptoms. Radiographic evidence indicated an osteolytic lesion in the lateral portion of one supraorbital ridge with involvement of the adjacent frontal squama, orbital roof, and zygomatic process of the frontal bone in all eight cases in which x-rays were taken. Local excision resulted in prompt recovery. Conservative operative approach and curettage were recommended. The etiology and pathogenesis remain unknown. It was suggested that these lesions may be related to histocytic granulomas of the Schüller-Christian type.

Shuster and Flynn [276] stated that cases of eosinophilic granuloma of the frontal bone are commonly referred to the otolaryngologist in view of the swelling in the orbital region and are frequently assumed to be of sinus origin.

Belal [277] reviewed the different types of meningioma which possibly can invade nasal sinuses, the nasal cavity, and the orbit.

Conley, Pack, and Trinidad [278] reported a case of extracranial meningioma involving the infratemporal and orbital regions. It was removed by a direct approach to the infratemporal space, which gave better exposure of this region than the usual scalp flap.

Midline Lethal Granuloma.—When generalized periarteritis is associated with a lethal midline facial granuloma, the syndrome is known as "Wegener's granuloma." When the disease process is associated with generalized necrotizing arteritis, varying degrees of infiltration are usually demonstrated in the parenchyma of the lung. In Geist and Mullen's [279] brief review of lethal granulomatous ulceration of the midline facial tissues, emphasis was placed on the roentgenological aspects.

Godman and Churg [280] presented the pathology of seven cases of a syndrome (Wegener's granulomatosis) presenting severe destructive lesions of the respiratory tract, arteritis, and nephritis. This disease is characterized by aggressive necrotizing granulomas of the respiratory tract, generalized angiitis, necrotizing glomerulitis and frequently disseminated granulomas.

Paterson [281] summarized the various theories on the etiology of Wegener's granuloma. There does not appear to be any specific treatment for this disease, but in his series the combined use of antibiotics and radiotherapy seemed to give the best results. Cortisone and corticotropin (ACTH) did not appear to be of any benefit in this disease.

Friedmann,[282] too, emphasized the association of malignant granuloma of the nose with necrotizing, vascular, and granulomatous lesions of other organs.

McCallum [283] described a case of fatal chronic sinusitis, nasal granuloma, and periarteritis nodosa. The pathological features correspond to the hypersensitivity angiitis as described by Zeek, rather than the classic

form of periarteritis nodosa. The allergic reaction has been held to be due to bacterial allergens.

To Glass [284] x-ray therapy seemed to be beneficial in cases of malignant granuloma of the nose and he recommended that it be used more vigorously and more often in these cases.

Ellis [285] and Howell [286] reported cases of malignant granuloma treated by radiotherapy with immediate good results.

Milner [287] reported the case of a woman, 41 years of age, who presented with granulomatous rhinitis and gingivitis, and who died in uremia. The case was unique in two respects: (1) the nasal and gingival lesions healed before death, and (2) no vascular or granulomatous lesions could be found in the lungs at necropsy.

Alexander,[288] Breckenridge and associates,[289] Bandler and Campbell,[290] Lane and Dodd,[291] and Kelly [292] presented cases of lethal idiopathic granulomas of the nose and face.

Howells [293] reported two cases of proptosis and granuloma of the orbit. In each, the orbit was exenterated and irradiation was administered after operation with complete relief for six months. Howells considered these as cases of so-called malignant granuloma.

Miscellaneous.—Zwiefach [294] in his review of the entire subject of rhinoscleroma pointed out that the disease affects not only the nose but other parts of the respiratory tract as well.

Folbre and collaborators [295] added a case of rhinoscleroma successfully treated with terramycin to the evidence that Klebsiella rhinoscleromatis (the von Frisch bacillus) is the causative agent of this disease.

Ottosson [296] presented a case of primary isolated amyloidosis of the nose with complete filling of both nasal cavities in an 86-year-old man.

Radiation treatment is the method of choice for carcinoma of the skin of the nose in all but exceptional cases, according to Delario.[297]

A malignant schwannoma of the tip of the nose developed in Conley's [298] patient with radiodermatitis.

Crawford and associates [299] reported seven cases of dermoid cyst of the nose treated at Duke Hospital. The characteristic features were described and differential diagnosis was discussed.

Goldie and Macbeth [300] presented two cases of midline nasal dermoid cysts.

Rao [301] described a cyst pushing forward the junction of the ala nasi with the upper lip. He preferred the name "nasolabial cyst." The lesion can be easily removed through a sublabial approach.

Van Eycken [302] reported a case of cyst of the nasal vestibule, and Doyle-Kelly [303] and Belal,[304] cases of so-called bleeding polypus of the nasal septum. When the latter are partially or improperly removed, they tend to recur; therefore some of them had been thought to be sarcomatous.

Jones [305] reported a case of leukoplakia of the septum in Kisselbach's area.

Ear and Mastoid

The clinical and pathological features in 94 instances of chondrodermatitis nodularis chronica helicus were considered by Newcomer.[306] The painful nodule represents essentially a vascularized organizing inflammatory focus within the cutis. The inflammatory nodule once established commonly extends to the perichondrium, but the auricular cartilage itself shows only degenerative changes incidental to aging. Contrary to the prevailing view, the degenerative changes do not appear to play any part in initiating the lesion or in its development. No method of treatment can be depended on to give consistently satisfactory results. Management consists of injections of procaine followed by conservative excision if symptoms are not relieved.

Malignant Lesions.—Cancer of the auricle located within 1 cm. of the external auditory meatus is a serious disease. Miller [307] favored treatment by radical removal of most of the auricle and the under-

lying mastoid cortex en bloc and modified radical mastoidectomy in chosen cases. This may seem radical esthetically because of the removal of most of the auricle, but it is not at all radical in the light of the high rate of failures.

Fredricks [308] surveyed 54 cases of malignant tumor of the external ear. He discussed the various types of surgical treatment for such lesions and stressed the difficulties of the radiotherapeutic treatment.

MacGibbon [309] and Bennett and Davis [310] reported cases of tumor of the external auditory canal, middle ear, and mastoid, and Friedmann and Radcliffe [311] and Sharnoff and associates,[312] instances of otosclerosis associated with multiple myeloma and malignant melanoma of the auditory canal.

Brunner [313] used the term "basal-cell carcinoma" in the broad sense, to include tumors which are not classified as squamous-cell carcinomas, in other words, tumors which are supposed to originate in the basilar layers of the epidermis and its appendages or in the salivary gland. In the case he reported, an adenocarcinoma caused paralysis of the facial nerve and involved the parotid, mastoid, and external auditory canal.

Although productive of early symptoms readily approachable for histological diagnosis and slow to spread beyond the confines of the temporal bone, cancer intrinsic in the middle ear has been a highly lethal disease. When extrinsic cancer has reached the middle ear and mastoid, aggressive and radical treatment still may save many patients. From the standpoint of long survival, Figi and Weisman [314] stated that management of chemodectoma in the middle ear with radiation therapy and conservative operative procedures has been satisfactory. They reported a study of 95 tumors of the middle ear and mastoid; 47 were intrinsic in the middle ear and mastoid; 26 were extrinsic, and, after a histopathological review in all cases, 22 tumors were classified as intrinsic chemodectomas.

The problem of curing cancer of the middle ear is concerned chiefly with adequate local removal, and this seemingly applies particularly to bone, as irradiation appears to be effective in controlling the soft-tissue invasion. Before treatment is begun in any case, a clear idea must be entertained of the possible directions of spread in the bone and a feasible means of excising these portions. Ellis and Pracy [315] stated that such excision is not an insurmountable technical problem, provided the problem is not presented suddenly in the course of an operation conceived in minor terms.

Brown [316] reported on seven patients afflicted with epidermoid carcinoma of the temporal region. The poor results of treatment were emphasized. The possibility of resection of the temporal bone followed by radiation was recommended for consideration.

Parsons and Lewis [317] described block excision for cancer involving the ear and mastoid. It was too early to evaluate results in their 11 cases with two deaths in the postoperative period. All but two of the patients had had some minor attempt at excision plus irradiation first. This experience has shown that block resection of the ear canal, middle ear, and mastoid is practical.

Bradley and Maxwell [318] reported on 54 patients with neoplasms involving the middle ear and mastoid treated at Ann Arbor, Michigan, during the 35 years from 1917 to 1953. It would seem doubtful from this study that chronic suppuration in the middle ear is a highly important factor in etiology of cancer of the middle ear and mastoid.

Twenty-seven cases of carcinoma of the middle ear and mastoid region were reviewed by Adams and Morrison.[319] In 18, chronic otitis media or a previous history of ear disease was present. The best results were obtained when the disease was at an early stage and still confined to the temporal bone. Surgical exploration and removal of the tumor followed by radium beam therapy

seemed to offer the best chance of survival. Five patients were alive and well five years after treatment.

According to Boland and Paterson,[320] their experience indicated that x-ray treatment holds considerable promise of curing cancer of the middle ear and mastoid and they hoped that future results would show a higher percentage of cures.

Lindahl[321] reported a study of 16 cases of cancer of the middle ear, and Lodge, Jones, and Smith[322] presented a classification of tumors of the temporal bone.

The majority of recorded cases of rhabdomyosarcoma have been in children and young adults. A distinct group arises in the head and neck, and has a predilection for the palate, middle ear, and orbit. Treatment, whether by excision or irradiation, is usually followed by recurrence at the primary site. Holman[323] reported a case of rhabdomyosarcoma of the region of the middle ear in a boy, aged 2 years, with a solitary metastatic lesion in the lung.

Jones[324] reported a case of carcinoma arising in a mastoid cavity. The patient had a right radical mastoidectomy one year prior to the onset of foul discharge and pain and the discovery of carcinoma. The patient had suffered from bilateral otorrhea for 30 years.

Cholesteatoma and Cyst.—According to Biber,[325] the so-called primary cholesteatoma of the external auditory canal does not exist, for a cholesteatoma is secondary in nature, mostly due to inflammation in the middle ear. In most cases the evidence will indicate more or less development of granulation tissue, which constitutes the place where the potentialities of two kinds of epithelium cross. The stronger epithelium proliferates abundantly, burying in the depth of the weaker one and thus providing the starting point for formation of epithelial islets.

Winderen and Zimmer[326] searched the records concerning 536 ears with chronic infection of the middle ear for those in which cholesteatomas in the middle ear and vicinity were found at operation. Their work dealt only with the so-called pseudocholesteatomas, or false cholesteatomas. Their report was divided into two parts, a clinical and a roentgenological section. In a patient with chronic suppurative otitis media, marginal perforation of the membrane, and aural discharge for 10 to 20 years, the risk of serious complications is sufficiently great to warrant surgical intervention. Cholesteatoma was demonstrated in 65% of the ears with chronic otitis media subjected to operation. This paper should be read in toto by students of the disease.

Meltzer[327] found cholesteatosis in 43 children 10 years old or younger. These cases afforded one example after another of diagnosis made too late to save the hearing. He stated that today, because antibiotics are used to control the exacerbations of chronic suppuration, cholesteatosis has an even better chance of going undiscovered along its destructive path, invading and damaging beyond repair structures vital to the child's hearing.

To Baron[328] the clinical evidence was overwhelming that in certain situations it is to the patient's advantage for the surgeon to preserve the matrix of the cholesteatoma. This is especially true in the atticoantral type of cholesteatoma with a sealed off middle ear. The matrix per se is not a dangerous structure. In certain circumstances the matrix may be removed: namely (1) to eradicate infection and (2) to eliminate evagination into pockets in the bone and to exenterate the underlying cells. The reasons for preserving the matrix when not contraindicated are (1) to aid in epithelization of the cavity, and (2) to conserve hearing.

In the absence of infection, moisture and retention of debris and crust, Lindsay[329] found no evidence that the matrix causes absorption of underlying bone.

Walsh[330] found considerable clinical evidence that the theory of pressure necrosis as a cause for bone erosion in cholesteatoma is untenable. Some evidence indicated

a chemical osteolytic process. In his opinion any surgical procedure which neglects removal of the cholesteatoma matrix is inadequate.

Lurie [331] noted that serious problems arise when cholesteatoma involves other areas than the middle ear and mastoid.

House [332] reported on the unusual phenomenon of apparent primary cholesteatoma occupying the area of the stapes. Since operation the patient has been free of the vertigo, which was apparently produced by pressure from the cholesteatoma in the area of the oval window.

Turnheim [333] reported a rather unusual case of cholesteatoma filling a mastoid cell that remained after radical mastoidectomy. The cholesteatoma expanded downward, and after breaking through the cortex of the tip of the mastoid bone, formed a cyst in the neck.

In Day's [334] case a probable cholesteatoma verum of the middle ear arose near the pyramidal eminence.

Brunner,[335] emphasizing diagnosis, presented three cases of cholesteatoma.

Richardson's [336] case of a cyst of the mastoid suggested that an intact eardrum does not always indicate a normal ear. Roentgenologic study of the mastoids should be made of patients with vague symptoms.

Eosinophilic Granuloma.—The clinical evidence suggests that the three diseases, eosinophilic granuloma, Letterer-Siwe disease, and the Hand-Schüller-Christian syndrome, are varieties of one disorder. Treatment by irradiation is reserved for the relief of pain and making patients more comfortable. Dawes [337] stated that prognosis should always be guarded, for the behavior of the lesion in any one individual patient cannot be foreseen.

Everberg [338] reported a case of histiocytosis simulating otitis with temporal osteitis. Biopsy showed eosinophilic granuloma of the temporal bone, and a biopsy of a lymph node showed Letterer-Siwe disease. Postmortem diagnosis was Hand-Schüller-Christian disease.

Chisolm [339] stated that Hand-Schüller-Christian's disease may simulate middle ear or mastoid suppuration in children. X-ray examinations in suspected cases should include the entire skull and other membranous bones.

Vaheri [340] reported another case of eosinophilic granuloma. The focus in the mastoid process initially caused external otitis and, later, symptoms resembling mastoiditis.

Hoxie [341] reported a case of eosinophilic granuloma occurring bilaterally in the temporal bones of an adult with a 12-year medical history.

Acoustic Neuroma.—The chances of a satisfactory outcome from operation on cases of acoustic neuroma are greatly increased by early diagnosis. Complete neurological examination, including caloric and audiometric testing, of all patients with perception deafness is hardly possible; but a carefully elicited history and an examination restricted to gait, stance, and the fifth, seventh, and eighth cranial nerves will usually suffice for one to pick out the cases in which full-scale otological investigation is necessary. The importance of the loudness balance test was emphasized by Elliott and McKissock.[342]

Brown, Love, and Adams [343] reviewed 150 Mayo Clinic cases of unilateral acoustic neurofibroma in which operation was performed and in which the diagnosis was confirmed by microscopic examination of tissue. On the basis of this study the following conclusions were drawn: 1. Any degree of nerve deafness with or without disturbed equilibrium in the presence of spontaneous nystagmus, decreased corneal sensitivity, atypical facial pain, paresthesias of the face or tongue (rarely the extremities), unexplained headache, facial weakness, visual disturbance, or ataxia is suggestive of an intracranial lesion and indicates that complete cochlear and labyrinthine evaluation should be carried out

and that roentgenologic examination of the head, examination of the ocular fundi and neurological consultation are in order. 2. The finding of signs suggestive of intracranial disease on the side opposite that of the deafness does not rule out the possibility of acoustic neurofibroma.

In a small percentage of cases of acoustic neurinoma, deafness is late in appearing. The occasional absence of clinical deafness even with large tumors is probably related to the pathogenesis of the growth. Jones and Kerr [344] stated that audiometric examination should be undertaken in all suspected cases. They reported three cases of acoustic neurinoma in which deafness did not develop until very late in the disease.

The early diagnosis of acoustic neurofibroma simplifies surgical removal, minimizes the amount of adjacent cranial nerve damage and reduces mortality. Unfortunately, in the majority of patients with tumors of the eighth nerve surgical removal of the tumor is delayed for want of early diagnosis. Morris [345] emphasized that the failure to obtain x-ray evidence of a tumor should not influence the clinical diagnosis nor delay surgical intervention if such a procedure seems indicated by appropriate clinical findings.

Denny [346] stated that a marked disproportion between the depression of acoustic and labyrinthine function seems to be an important early finding in acoustic neuroma. He stressed the importance of tomography in the x-ray examination of the internal auditory meatus.

Bengochea and colleagues [347] reported on a meningioma of the internal auditory meatus in a 3-year-old child.

Craig, Dodge, and Ross [348] reported two cases of acoustic neuroma in children, ages 11 and 14 years.

Tumor of the Glomus Jugulare.—In 1942 the Department of Pathology of Mount Sinai Hospital received for study a polyp removed from the middle ear, which, on histological examination, was classified by Otoni as a carotid-body-like tumor. As origin for this tumor, he suggested a structure described in 1941 by Guild, the jugular glomus, located in the adventitia of the uppermost part of the internal jugular vein at the base of the skull as well as along the tympanic nerve within the middle ear. The surgeon, Dr. H. Rosenwasser, reported the case in 1945, and directed the attention of otologists and pathologists to this new entity. Zak [349] preferred to call the tumor Otoni's tumor. The discovery by Otoni has clarified a great number of variously labeled growths of the petrous bone and has shed light on mediastinal, orbital, ophthalmic, cranial, meningeal, and cerebral neoplasms. Thus the cerebellar hemangioblastoma and similar supratentorial neoplasms are classified as of glomic origin, as is their retinal counterpart. Van Hippel-Lindau's disease is considered a multicentric glomic neoplasm.

Guild [350] sought for the glomus jugulare in the histological sections of 88 human temporal bones. The average number of jugular bodies found was slightly less than three per ear. In none of the locations in which the glomus jugulare may be present is the structure of constant occurrence. The differences in clinical symptoms that have been reported for patients with tumors of the glomus jugulare, also the differences in apparent site of origin found at operations for such tumors, can all be satisfactorily explained by the differences in the observed locations of the normal glomus jugulare.

Siekert [351] studied 33 patients with tumors of the glomus jugulare from the neurological standpoint. Involvement of the acoustic nerve was noted in 27. Other neurological involvement was also present in 14 patients. Involvement of the 3d through the 12th cranial nerves, except for the 4th, and bruit, ataxia, nystagmus, Horner's syndrome, and exophthalmos were present in various cases.

Crue and associates [352] presented two cases of neoplasm of the posterior fossa involving the jugular foramen. One was a tumor of the glomus jugulare, the other a

neurofibroma. Involvement of all three of the nerves passing through the jugular foramen (9th, 10th, and 11th cranial nerves) occurred. Ageusia, dysphonia, dysphagia, and weakness of the neck and shoulder were present.

Huppler, McBean and Parkhill[353] reported a case of chemodectoma of the middle ear with paralysis of the vocal cords as a presenting symptom.

Chambers[354] presented a case of glomus jugulare which illustrated the difficulty of differential diagnosis when no mass presents in the ear.

Burman[355] described the known components of the chemoreceptor system with respect to their structure, development, innervation and function. The structure and behavior of the chemodectoma, the tumor of the chemoreceptor system, were discussed.

Holesh[356] stated that tomography in the anteroposterior projection is much 'more reliable than the conventional views of the petrous bone and should be employed whenever tumors of the glomus jugulare are suspected. Cerebral arteriography is not indicated if, as so often happens, the diagnosis can be made on the clinical and tomographic findings. Tomography will show destruction of the lower portion of the petrous pyramid, the ridge remaining as a more or less intact shell. This type of erosion peculiar to glomus tumor can be demonstrated satisfactorily only by tomography.

Hooper[357] noticed that the radiographic changes of tumors of the glomus jugulare most frequently seen are sclerosis of the mastoid region, enlargement of the jugular foramen and erosion of the petrous portion of the temporal bone. Angiograms of the external carotid artery were most helpful in diagnosis.

Williams and associates[358] treated 13 patients with chemodectoma of the glomus jugulare with a combination of surgical procedures and radiation or with radiation alone. It seems probable that in the future uncomplicated chemodectomas may be best treated by radiation alone.

Stewart, Ogilvie, and Sammon[359] stated that the choice of treatment of tumor of the glomus jugulare lies in a limited surgical removal either by simple removal of the growth through the external meatus or by radical mastoid, combined with radiotherapy. The radiotherapeutic action on these tumors would appear to be mainly on the vascular spaces, thereby causing a reduction in the blood supply and consequent shrinkage in the bulk of the growth and not as one might suppose by a direct action on the tumor cells themselves.

Shambaugh[360] stated that since these tumors (glomus jugulare) are not radiosensitive early surgical removal is advisable. He described the technique of hypotympanotomy for removal of early glomus jugulare tumors confined to the hypotympanic and tympanic cavities.

Rosenwasser[361] presented an excellent review of glomus jugularis tumors of the middle ear, carotid body tumors, tympanic body tumors and nonchromaffin paragangliomas.

Riggs and Tamari[362] stated that during the last nine years a total of 75 cases of nonchromaffin paraganglioma in the temporal bone have been reported which they considered reliable from their clinical aspects and tissue examination. Thirty-seven more cases were added by Flynn, Smith and Luongo,[363] Strong,[364] Pérez-Toledo and Taveras,[365] Davol,[366] Blomqvist, Lindwall and deMaré,[367] Paulsen,[368] Brown,[369] Lane,[370] Towson and McNelis,[371] Amman,[372] Glanville,[373] Riggs and Tamari,[374] Cleary[375] and Rao.[376]

Schade[377] examined seven tumors of the glomus jugulare and five of the glomus caroticum, and their histological characteristics were found to be identical.

Garvey and Claudon[378] reported a case of nonchromaffin paraganglioma of the middle ear and retroperitoneal portion of the abdomen.

McNeill and Milner [379] reported their experience with bilateral tumor of the glomus jugulare in a woman aged 70 years. The mastoid was explored, but because of torrential hemorrhage the procedure was abandoned.

Miscellaneous.—Seltzer [380] and Kline and Pearce [381] reported cases of osteoma of the mastoid and auditory canal. Even a fairly large osteoma does not demand surgical intervention when it is not causing symptoms. If there is impaired hearing, persistent fullness in the ear or pain, removal may be indicated. The ease with which the tumor can be removed depends on its size and the width of its pedicle.

McKibben and Sheehy [382] reported a case of tuberculosis of the middle ear and nasal passages, associated with facial palsy in an apparently healthy pregnant young woman.

Benign chondroblastoma has been found chiefly in the extremities near the epiphyseal centers. Denko and Krauel [383] found such a tumor in the temporal bone. Their case is the only one reported to date in which a chondroblastoma has been found in the flat bones or skull. Conservative treatment, consisting of surgical curettage, is the recommended procedure.

Jaws

Carcinoma and Sarcoma.—One hundred fourteen radical combined operations for cancer of the head and neck were performed at the Memorial Hospital, New York, in 1950. According to Friedman and Ewing,[384] the operative mortality was only 0.88%. This study underscores the fact that extensive radical operation can be performed with a remarkably low mortality and morbidity even on elderly and debilitated patients. The specific operation referred to consists of radical dissection of the nodes of the neck, resection of a portion of the mandible, and excision of the intraoral primary lesion.

A logical plan based on anatomical and surgical principles and a knowledge of the growth habits of cancer should be used for determining the proper extent of mandibular resection without creating needless deformity. Byars [385,386] stated that bone must be considered to be invaded by oral cancer when a malignant process has penetrated its thin mucosal covering. When the mandible is involved, the surgeon may select one of three procedures, depending on the extent of the disease. The basic consideration has to do with whether or not the inferior dental canal with its vessels, nerves, and lymph vessels has been entered by cancer cells. Proper attention to mandibular support usually obviates the necessity for tracheotomy. The purpose of internal support with wire prosthesis after resection of the mandible is to make the patient's postoperative course as easy as possible by enabling him to eat and breathe easier. In most instances, it is a temporary splint which is removed after two to six weeks.

Pollack [387] reported the early results from a combined operation in a small series of cases of intraoral cancer. Immediate plastic reconstruction of the mandible was not as satisfactory from the standpoint of curing the cancer as reconstruction at a later date.

Stewart and Stafne [388] found tumor cells involving the dental pulp in only one of 39 cases of malignant tumors of the oral cavity. The incidence of tumor involvement of the dental pulp by malignant tumors of the oral cavity appears to be low.

Castigliano and Rominger [389] reviewed the literature from 1902 to 1953 and found a total of 176 cases of metastatic malignant disease involving the jaws. The primary lesions most frequently accounting for metastasis to the jaw were carcinoma of the thyroid, hypernephroma, carcinoma of the breast, lung, and prostate. The majority of reported lesions were osteoclastic. If, for no other reason, the possibility of a metastatic malignant growth involving the jaws should be emphasized in the diagnosis of neoplasms in that location in order to spare a significant number of patients with occult malignant lesions the tragedy of useless disfiguring radical surgery.

Salman and Langel[390] reported seven cases of metastatic lesions to the jaws, two to the breast, one from the kidney, one to the uterus, one from the prostate and one from the lung.

Wildermuth and Cantril[391] stated that with modern antibiotics and meticulous care, the teeth can be extracted when and if they become painful and carious after treatment with irradiation. Irradiation before reconstruction of the alveolar ridge of the mandible, after prophylactic extraction of the teeth in the field of irradiation, results in permanently delayed remodeling of the mandible and a persistent threat to the integrity of the overlying protective gingival mucosa.

When there is carcinomatous invasion of the jaw bones, destruction of bone occurs most commonly at the alveolus and usually is apparent as a V-shaped area of erosion which is rather characteristic. Sherman and Chu[392] stated that the edges may be either distinct, ill-defined, or at times slightly ragged. If teeth are present within the tumor area, destruction of the lamina dura usually is evident.

In attempting to diagnose osteogenic sarcoma of the jaws roentgenologically Sherman and Melamed[393] evolved a broad grouping consisting of three principal types: the sclerosing, the lytic, and the mixed. Radiographically, the sclerosing osteogenic sarcoma presents as its main feature a predominance of dense structureless eburnated bone. In the lytic form destruction predominates, and usually no density may be expected. The mixed type presents approximately equal amounts of destruction and production. The radiographic appearance of the typical well-developed sclerosing osteogenic sarcoma is just about pathognomonic. The other two types cannot be identified (that is, no specific roentgen diagnosis can be made).

According to Gerry and Williams,[394] the commonest site of osseous involvement of the head by reticulum-cell sarcoma is the mandible. They presented two such cases. Only six cases of mandibular involvement have been previously reported in the literature.

Stuteville[395] reported a case of fibrosarcoma of the mandible.

Paterson[396] described a case of chondrosarcoma of the maxilla. The pathology is summarized, together with the various theories of origin of cartilage tumors in bone.

Link[397] reported a case of chondrosarcoma of the maxilla which he said was the ninth case to be reported.

Chondroma of the jaws is a far more sinister tumor than most of the textbooks would have us believe. The treatment must be radical. It is best to suspect all cartilage tumors as being at least potentially malignant and to treat them accordingly. Blum[398] reported three cases.

Adamantinoma.—Ameloblastomas or adamantinomas are rare epithelial tumors which usually have one of two patterns: (1) a follicular type in which the tumor elements form masses resembling enamel organs, and (2) a plexiform type consisting of large branching masses of stellate epithelial cells. About 1036 cases have been reported. Small and Waldron[399] stated that about 1% of the cysts and tumors of the jaws may be expected to be ameloblastomas and that 81% of the ameloblastomas are found in the mandible.

Twenty-nine patients with adamantinoma of the mandible were admitted to the Presbyterian Hospital, New York City, during the past 30 years. Rankow and Hickey[400] noted 91% recurrence after local curettement for these adamantinomas. By contrast, no recurrences have followed hemisectomy of the mandible.

Thirty-six patients with adamantinoma were admitted in 15 years to Duke Hospital, Durham, N. C. Treatment of choice for ameloblastoma of the mandible wherever possible is resection and immediate autogenous grafting, according to Georgiade and associates.[401] A number of patients, however, in whom disarticulation was per-

formed because of involvement of the angle and ramus of the mandible did not desire reconstructive surgery because of the minimal disfigurement and loss of mandibular function. Eighty per cent of the patients were less than 35 years old.

Campbell [402] examined a series of 19 adamantinomas. Large granular cells, resembling those seen in congenital epulis and granular cell myoblastoma, were found in two adamantinomas. No definite conclusions could be made regarding the pathogenesis or significance of this observation.

Sherman and Caumartin [403] stated that in their opinion familiarity with the appearance and behavior of adamantinoma and careful analysis of its roentgen features will lead to an accurate diagnosis by the roentgenologist in the great majority of instances.

Butler and Harrigan [404] recommended adequate local excision by an extraoral route for the early adamantinoma. In such cases continuity of bone can be retained, and cautery is not necessary. Large extensive tumors require resection. Primary grafting, whether metallic or bone, should be avoided.

Holdsworth and Rowe [405] presented a case of adamantinoma which was treated by resection and immediate replacement by a prefabricated graft of tantalum and acrylic resin. The condition after two years seemed satisfactory.

Bonta and James,[406] Hinds, Pleasants and Snyder,[407] Fisher [408] and Flynn and Tinning [409] presented cases of adamantinomas. Villa [410] presented a case of ameloblastic sarcoma of the mandible.

Haas and Ritter [411] and Spaulding [412] reported single cases of adamantinoma of the maxilla.

Fibrous Dysplasia and Ossifying Fibroma.—Fibrous dysplasia frequently involves more than one facial bone. Most often the maxilla is involved. In view of the benign nature of fibrous dysplasia, massive resection of the bone is seldom justified. Kanthak, Hamm, and Yarn [413] stated that treatment, if any, should be directed toward restoring contour and function.

Thoma [414] considered it difficult to distinguish between fibrous dysplasia and ossifying fibroma by pathological examination. In fibrous dysplasia the entire bone or a great part of it may be involved, and when it affects the middle of the face, it tends to involve adjacent bones. Ossifying fibroma forms a tumor mass which encroaches on adjacent normal bone.

Gold's [415] classification of fibrous dysplasia of the jaws, both osseous and odontogenic, was based on the histological interrelationships of its stages; he described and discussed osseous dysplasia and analyzed and correlated the clinical and histological findings in a series of cases.

McDonald and Shafer [416] reported a case of fibrous dysplasia of the jaw in a 5-year-old girl. Their case was similar to others reported as "cherubism," a familial multilocular cystic disease of the jaws. One outstanding similarity in all cases is the absence of a number of permanent teeth. The dystrophy probably is related to odontogeny, and thus is not unlike a hamartoma. Fibrous dysplasia of the jaws, which may be congenital, apparently progresses rapidly during early childhood but later may remain static or even show evidence of slight regression.

Georgiade and associates [417] presented 34 cases of ossifying fibroma. Complete visualization and removal of extensive ossifying fibromas of the facial bones can be accomplished by means of an external approach with minimal postoperative deformity.

Miscellaneous Lesions.—Perin [418] and Bruce [419] reported a case of osteofibroma of the maxilla and a case of odontogenic fibroma of the mandible respectively.

Myxoma is a soft, gelatinous tumor which histologically consists of branching stellate cells in a loose mucoid basophilic stroma, which may be finely fibrillated or granular. The tissue resembles embryonic mesenchyme and has its prototype in Wharton's jelly of the umbilical cord. It may be impossible to distinguish between benign and malignant

forms. Brewer and Johnston [420] stated that central myxoma of the jaw occurs most commonly in the second or third decades and leads to a painful swelling. It is usually associated with unerupted or congenitally missing teeth. They reported such a case.

It is probably safe to assume that most lesions described as giant-cell tumors of the jaws are not true neoplasms and only mimic the giant-cell tumor of bone. Waldron [421] was of the opinion that these growths may properly be designated as giant-cell lesions, giant-cell nodes or giant-cell reparative granulomas to emphasize their benign, non-neoplastic nature.

Pleasants and MacComb [422] reported a case of multiple central giant-cell tumors of the jaws, one occurring in the mandible and another in the maxilla of the same patient.

Foss, Dockerty and Good [423] presented a case of osteoid osteoma of the mandible. No other case of such an authenticated lesion could be found in the literature. Osteoid osteoma of the mandible must be extremely rare. This is the only case in which osteoid osteoma of the mandible has been seen at the Mayo Clinic.

Nelson [424] reported a classic osteoid osteoma in an unusual location, the maxilla.

Cornell and Vargas [425] and Bruce [426] each presented a case of neurofibroma of the mandible.

Mark [427] reported a case of central fibroma of the mandible.

Hinds, Pleasants, and Bell,[428] and Davis and Havens [429] reported cases of plasma-cell myeloma of the mandible.

Albano, Aquino and Modi [430] reported a case of solitary eosinophilic granuloma of the mandible.

Szerlip [431] reported a case of cystic muco-epidermoid tumor of the mandible.

Mathieson [432] described a case of fibro-cystic salivary tumors of the mandible. Origin from undifferentiated submandibular salivary gland tissue was postulated.

Epulis gravidae is a tumor of the gingivae of pregnant women. The tumor occurs in 2% of all pregnancies. Many of the small ones regress on termination of pregnancy. Goodridge [433] stated that if signs of tumor formation have not appeared in four and one-half months, it is unlikely that there will be any later growth.

The torus palatinus is a bony elevation along the median suture of the hard palate. It is often not noticed until it becomes superficially ulcerated; then it produces mental anxiety and is mistakenly identified as a malignant tumor. James and Walter [434] emphasized that it should not be treated unless it is causing the patient marked difficulties. It has never been reported as the source of a malignant neoplasm.

Huebsch and Stephenson [435] reported two instances of benign osteogenic enlargement or overgrowth of the condyloid process, occurring after puberty. The treatment of choice in benign unilateral local osteogenic tumors of the mandibular condyloid process appears to be surgical removal.

Whinery [436] suggested the term "progressive bone cavity" to replace the term "traumatic bone cyst" of the mandible. The lesion neither has a sac-like definite wall nor does it necessarily contain fluid. It is a hollow place within the bone. Etiology is unknown.

Blum [437] and Jacobs [438] presented cases of progressive mandibular bone cavities, and Caldwell and Thompson,[439] a case of bilateral multilocular follicular cysts of the maxilla and mandible.

Hayton-Williams [440] reported a case of arteriovenous fistula or aneurysm of the mandible.

Retinal anlage tumor is a benign, pigmented tumor of childhood. Caldwell, Ernst and Thompson [441] stated that a review of the literature revealed involvement of the maxilla in seven cases, the skull in one, and the mandible in two.

Trabue and Martin [442] reported a case of retinal anlage of the maxilla in a 10-week-old girl.

The name "congenital epulis" has been introduced into the medical literature (1871) to designate a soft growth observed on the

gingiva of the maxilla of a newborn infant. The term "epulis" has a topographic generic meaning and is used to denote different growths in the gingiva. The name "congenital epulis" is poorly chosen since this tumor differs fundamentally from the so-called common epulis which is an inflammatory hyperplasia of the gingival mucosa. Bauer and Bauer [443] stated that the so-called congenital epulis is a granular cell fibroblastoma.

According to Bhaskar and Akamine [444] a review of the previously reported cases of congenital epulis shows that this tumor involves predominantly the maxillary alveolar mucosa of the newborn female.

Hankey [445] reported a case of congenital epulis.

Miles [446] presented a case of fibroma of the maxilla.

Quinn and Fullmer [447] presented a rare tumor, central ossifying myxofibroma of the maxilla, while Villa [448] presented a case of fibrous dysplasia of the upper jaw which was undergoing myxomatous degeneration.

Hovnanian [449] presented two cases of myxoma of the maxilla, bringing the number reported thus far to nine. The radical removal of the maxilla seems to be the only rational approach to the problem in the advanced stage of the disease.

Christiansen [450] reported a case of adenocarcinoma of the right upper jaw.

Ambrecht and Waterman [451] reported a case of primary neuroblastoma of the maxilla.

Palate and Uvula

Heflich and Volk [452] reported a case of cylindroma of the palate.

Rao [453] reported a case of teratoma in the palate in a newborn child.

Sherman and Calman [454] presented a case of hemangioendothelioma of the palate. This was excised and the wound closed with a split-skin graft held in place with a denture.

Gridly [455] reported a case of lipoma of the soft palate.

Maguda and Bowers [456] reported seven cases of primary carcinoma of the uvula. In four of these cases, metastatic involvement to the cervical lymph nodes was present on admission.

Pollack [457] described the surgical technique for removal of an extraosseous adamantinoma in a 56-year-old man. The lesion seemed to be attached to the soft palate and left buccal cheek and had been present since birth.

(To be concluded)

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CORRECTION

In the Editorial Note by Dr. Ernest M. Seydell to the article "Voice and Breathing: A Report of Some New Concepts," by Esti D. Freud, Ph.D. (January issue, p. 1), the neurochronaxic theory of Husson was incorrectly called the myeloelastic theory.

Abstracts from Current Literature

Ear

PREDICTING HEARING LOSS FOR SPEECH FROM PURE TONE AUDIOGRAMS. ROBERT R. QUIGGLE; ARAM GIORIG; JAMES H. DELK, and ANNE B. SUMMERFIELD, Laryngoscope 67:1-15 (Jan.) 1957.

An attempt is made to correlate the pure-tone audiogram with the speech-reception threshold. It is pointed out that such a speech test is a threshold and not a conversational level test. Furthermore, the words used, the spondees, are not wholly representative of everyday speech. However, a correlation between the pure-tone audiogram and the speech-reception threshold is a start toward a correlation with actual speech.

An equation is developed using the tones 500, 1000, and 1500 d. v., each weighted as is necessary. It comes out viz., x_s (the predicted spondee speech-reception threshold) $=6.92+0.22x_{500}+0.35x_{1000}+0.2x_{1500}$ (x being the pure-tone thresholds). The equation, in two-thirds of the sample group used (in 20- to 29-year-old subjects) is accurate within 5 db. In the other third, there is apt to be a slightly greater error. Likewise there was greater error when applied. The formula was applied to random samples of older men.

HITSCHLER, Philadelphia.

DISCUSSION ON RECURRENT OTITIS MEDIA IN CHILDREN. LIONEL TAYLOR ET AL., Proc. Roy. Soc. Med. 50:405 (June) 1957.

This session was held jointly with the Section on General Practice, Chairman, Philip J. Jory, D.S.O., F.R.C.S., on Feb. 1, 1957. Causes (Table 1): 1. Eruption of teeth; 2. enlarged adenoids; 3. persistent perforation of tympanic membrane; 4. latent mastoid infection; 5. lymphatic hyperplasia; 6. idiopathic. . . . "The relation between teething and otitis media is undisputed" . . . but the "mechanics," or rationale of the condition, is not yet clear. If recurrence of the otitis comes on with every severe cold, removal of adenoids is indicated. There is often an associated acute maxillary antritis. . . . If the drum ruptures and a persistent large hole is residual, bathing becomes hazardous, lest water entering the middle ear may start a new invasion of bacteria. Even syringing such an ear for removal of wax may reproduce the old condition. However, some perforations can be closed by myringoplasty. Latent infections are common. The bacterial invasion lies dormant, only to flare up again in a few days or weeks. Inadequate use of antibiotics may be basic. Two such cases are reported in brief, which were cleared only after mastoidectomy. . . . Removal of adenoids in the very young may be followed by an increase of lymphoid tissue in and around the tubal orifice. Since this may not be accessible to surgery, radiotherapy may be in order. . . . In the idiopathic group, a plausible cause of recurrence may be Eustachian tubes which are "too open" and hence subject to repeated invasions. About the age of 14, these patients improve.

The discussion was continued by Dr. I. Friedmann, clinical pathologist, who dwelt on the over-all incidence and kinds of bacteria found in over 2000 patients: Staphylococcus, Streptococcus, Escherichia coli, Bacillus proteus, Pseudomonas pyocyanea, and "mixed types" in that order. Gram-positive cocci predominated; there were no penicillin-resistant strains among 669 cases with S. pyogenes present. . . . "In the writer's opinion, penicillin has remained the antibiotic of choice". . . . A shift from Gram-positive to Gram-negative bacilli "is characteristic of the bacteriology of otitis media." Treatment of chronic otitis media is largely surgical because of the marked changes in tissue structure. Four illustrations are given showing the results of work with guinea pigs, after injection of various bacteria into the middle ear.

Dr. G. F. Abercrombie continued with case reports on 82 patients with 115 attacks of otitis. The usual management was followed as to myringotomy and mastoidectomy. "At the end of this series, no case . . . had been allowed to become chronic, and no patient was left with a running ear. . . . Mastoiditis, like puerperal fever, seems to be a thing of the past."

Dr. David Wheatley said that otitis media has changed almost overnight from a surgical to a medical condition, and the main object is to annihilate the infection at the earliest possible moment. Most Staphylococcus infections are sensitive to penicillin. A synergistic

combination of penicillin and a sulfonamide is advocated. When allergy is present, one may be baffled. Antihistamines are given both locally and orally with a generally good effect. To eradicate Proteus was often difficult.

Further discussions were carried on by Mr. S. R. Mawson, Mr. A. G. Tressider, Dr. I. Roodyn, Mr. S. W. Allinson, Dr. G. O. Barber, Mr. F. C. W. Capps, Mr. G. H. Bateman, and Mr. J. F. Lipscomb. Dr. R. Goulding could not praise antibiotics so fully as other speakers. Maybe there is "a change on the natural history of the disease." Dr. L. W. Batten thought "early incision is bad. One should wait until pus has surely formed. Nothing is gained by putting a knife in too soon. Pain is seldom relieved, and nowadays "pus is not often there."

Mr. J. W. S. Lindahl thought that there are a number of "hard core cases that go on and on," but the majority clear up under proper care. Removal of adenoids brought improvement in many cases.

Mr. Lionel Taylor reemphasized Dr. Barber's mention of measles as a potent cause of otitis. . . . Dr. D. Wheatley was a little disturbed by a speaker who chose to wait for a perforation. Nowadays antibiotics would be given at once, and the drum would be incised when ready, but it often cleared without this surgery.

Chairman Jory in closing said that the evidence was in favor of antibiotics in ear disease, but the dosage must be adequate.

I. W. Voorhees, New York.

One Hundred Operations for Aplasia of the Ear with Imperforate External Auditory Canal. Dr. Marcel Ombredanne, Acta oto-rino-laring. ibero-am. 8:315, 1957.

The author analyzes his results in 111 cases of aplasia of the ear. Aplasia may involve not only the external, middle, and inner ears, but also the ossicles and the tympanic cavity. Aplasia may be bilateral or unilateral. Unilateral surgery should be performed if the opposite ear is not normal in appearance or function, or for psychogenic reasons. Surgery is not performed if the patient is less than 5 years of age.

The first stage of the operation is the construction or reconstruction of the auricle. The extent of surgery depends upon the amount of deformity present. Skin flaps and various types of cutaneocartilaginous grafts are fashioned to reconstruct a presentable auricle.

The second stage is the reconstruction of the auditory canal. The procedure involves a mastoidectomy, an exposure of the external horizontal canal, with a fenestration, and finally a plastic operation for the new canal.

The results in 111 cases were (1) good in 76 cases (68%); (2) fair in 26 cases (23%), and (3) poor in 9 cases (18%). The results were evaluated primarily on the basis of improvement in hearing as tested both by pure-tone and speech audiometry.

Among the postoperative complications there were 12 cases of facial paralysis (8 cleared up from 48 hours to 3 weeks postoperatively), meningitis, serous and suppurative labyrinthitis.

In conclusion, the author believes that the deafness which accompanies aplasia of the ear can be restored to a very useful level, provided the malformations do not involve the stapes of the semicircular canals.

Persky, Philadelphia.

The Direct Application of Ultrasonic Waves to the Vestibular Apparatus in the Treatment of Ménière's Disease. Michele Arslan, Acta oto-rino-laring. ibero-am. 8:358, 1957.

The author performs a simple mastoidectomy with an exposure of the external semicircular canal. An ultrasonic apparatus with a special applicator is applied to the semicircular canal. There is an exaggerated nystagmus after two or three minutes which is followed by a paralytic form. The author records excellent results with this form of therapy not only for the nystagmus, but also for the relief of tinnitus, and in many cases an improvement in hearing.

Exact details of the technique are not available in the article.

Persky, Philadelphia.

Larynx

THE DIMENSIONAL INTERRELATIONSHIPS OF THE MAJOR COMPONENTS OF THE HUMAN TRACHEOBRONCHIAL TREE. J. E. JESSEPH and K. A. MERENDINO, Surg. Gynec. & Obst. 105:210 (Aug.) 1957.

This study was instituted principally to determine whether or not the dimensions of the component divisions of the tracheobronchial tree have any constancy of relationship one to another. An analysis of 57 bodies (21 male and 36 female) was based upon fresh autopsy studies. In 21 male bodies there was found an average tracheal diameter of 2.2 cm., with a range from 1.5 to 2.7 cm. In the 26 female bodies, the average diameter was found to be 1.7 cm., with a range from 1.3 to 2.5 cm.

The average diameter of the right main bronchi in the male bodies was 1.7 cm., in the female bodies 1.5 cm. The diameter of the left main bronchus in males averaged 1.5 cm., in the female 1.2 cm.

The bronchial lengths averaged as follows: males: right main bronchus, 2.3 cm.; left main bronchus, 5.4 cm.; females: right main bronchus, 2.1 cm.; left main bronchus, 5.0 cm.

It is stated that the sum of the cross-sectional areas of the two main bronchi is approximately equal to the cross-sectional area of the trachea. This is not, however, a precise linear relationship.

The present study was intended to define some of the interrelationships of the normal human tracheobronchial system, so as to provide a sounder basis for certain surgical procedures. It was anticipated that planned excisions and reconstruction of major bronchi might thereby be facilitated.

FRIEDBERG, Chicago.

VASCULAR ANOMALIES OF THE RETRO-INFRAHYOID (PRETRACHEAL) SPACE AND THEIR IMPORTANCE IN TRACHEOTOMY. E. W. SCHELDRUP, Surg. Gynec. & Obst. 105:327 (Sept.) 1957.

In calling attention to the broadened indications for tracheotomy and the increasing number of these operations, the author reviews the previously published incidence of postoperative hemorrhages and cites figures estimating their occurrence in from 0.5 to 8.5% of cases. Many of these were fatal and were due to vascular abnormalities of the region.

It is suggested that the pretracheal space traversed in operations in this area should, with greater significance, be called the "upward extension of the superior mediastinum in the neck." The vascular structures which normally occupy the superior mediastinum may abnormally come to lie in this upward extension in the neck and therefore immediately in front of the trachea.

Attention is called to a large venous plexus from the inferior thyroid vein which may obscure the infraisthmic portion of the trachea, and to the thyroidea ima artery which may ascend to the isthmus.

Among the pretracheal vascular anomalies described are those affecting the left innominate vein, the innominate artery, the arch of the aorta, and the right common carotid artery. Examples are cited of these vessels being found in the lower cervical area directly over the trachea.

Posttracheal vascular anomalies involving the right subclavian artery are commoner than has hitherto been appreciated. The author cites 21 instances of the passage of this vessel between the trachea and the esophagus, a condition which would make accidental incision into the posterior tracheal wall a matter of considerable hazard.

The author takes issue with Jackson's blind two-stepped finger-guided operation and "feel" technique, and, though he does not question its swiftness, he does cast some doubt as to its being the safest method. He urges a thorough acquaintance with the anatomic details of the area and with the normally and abnormally placed blood vessels which one may encounter in the region.

FRIEDBERG, Chicago.

SQUAMOUS CELL CARCINOMA OF THE ESOPHAGUS. R. W. POSTLETHWAIT; W. D. SEALY; JOHN R. EMLET, and JOSEPH J. ZAVERTNIK, Surg. Gynec. & Obst. 105:465 (Oct.) 1957.

The incidence, symptomatology, and diagnostic findings of 253 patients with carcinoma of the esophagus have been analyzed. In the group admitted since January, 1942, operability was 58% and resectability 21%, both being considerably higher in tumors of the lower third of

the esophagus. All patients had squamous-cell carcinoma except one with adenocarcinoma of the middle third of the esophagus. Invasion of the mediastinal structures was the most frequent cause for nonresectability in middle-third lesions and lymph-node metastases in the lower-third lesions. The postoperative mortality rate after definitive resection was 25%. Five patients with squamous-cell carcinoma of the esophagus survived over five years and were living without evidence of recurrence up to eight years and five months.

AUTHORS' SUMMARY.

POSTANESTHETIC LARYNGEAL GRANULOMA. BOCA DE VINALS and J. PERELLO, Acta oto-rino-laring.-ibero-am. 8:370, 1957.

The authors present a case of bilateral granuloma of the vocal cords. This followed an intubation anesthesia. This was removed by indirect laryngoscopy, and the patient made an uneventful recovery.

The granuloma was undoubtedly due to trauma of the tube. They recommend vocal rest after intubation and in some cases vocal reeducation.

PERSKY, Philadelphia.

Nose

EIGHTEEN OPERATIONS FOR ATROPHIC RHINITIS. L. DEGELS, Acta oto-rhino-laryng. belg. 11: 61, 1957.

The author presents a series of 15 cases of atrophic rhinitis in which he uses a polyethylene implant submucous on the septum. He has modified his previous procedure by the molding of the plastic material into small pellets of various sizes. With this modification, there is a more uniform narrowing of the intranasal spaces, less absorption, and a greater permanency for the "take" of the implant. The cases are described in detail and photographs in color are presented to illustrate the postoperative results.

PERSKY, Philadelphia.

Pharynx

THE PROBLEM OF RECURRING ADENOIDS: AN EDITORIAL, Eye, Ear, Nose & Throat Month. 36:232 (April) 1957.

The management of recurring adenoids remains a problem. Adenoids may recur even after the most careful and thoroughly performed operative procedure. Reoperation often fails to solve the problem of adenoid recurrence. A solution to this problem has been sought in the field of radiation. The use of radium irradiation in the nasopharynx as originally advocated by Crowe and his associates has been a subject of much controversy. The potential hazard of radium, not only to the patient but also to the physician, is not unimportant. No physician should utilize this therapy without adequate training. A lack of qualification on the part of a surgeon in the performance of a special operation does not invalidate the operation. Likewise, radium therapy of the nasopharynx should not be condemned because of hazard due to a lack of experience in the administering of radium therapy or in the selection of patients for this treatment.

Constructive criticism should be directed to the need of more adequate training by the otolaryngologist in the application of radium without minimizing the dangers of irradiation. Irradiation therapy of the nasopharynx should not be a routine procedure or a routine part of an operative procedure. Clinical improvement follows the use of this therapy only if the adenoid tissue is the offending agent either by mechanical obstruction or by chronic infection. Irradiation has only one purpose, that of removing lymphoid tissue.

JENNES, Waterbury, Conn.

WALDEYER'S RING AND ITS PROBLEMS. CH. HENSCHEL, Acta oto-rhino-laryng. belg. 11:129, 1957.

Since Waldeyer's ring is essentially a part of the lymphoid system, there is presented a review of its histology, physiology, radiobiology, and immunology. The role of the reticulum cells, and the various factors influencing hypertrophy and atrophy and their relation to the adrenal cortex are discussed.

The author believes that there is a definite difference in the problems involving the adenoids and the tonsils (either palatine, pharyngeal, or lingual.) He discusses the role of pharyngeal lymphoid hypertrophy. This has little relation to the size of the tonsils or adenoids, except

in cases where this hypertrophy causes obstruction to the throat or the Eustachian tube orifice.

The Waldeyer ring either in toto or any portion (tonsils or adenoids) may act as a source of focal infection and thus be associated with many general diseases.

Treatment is discussed on the basis of general, local, surgical, and radiation. General treatment is concerned primarily with metabolic disturbances. Here the vitamins, hematinics, and correction of faulty diets are in order. The antibiotics are of value only in the acute infections such as acute tonsillitis. However, considerable attention should be placed on the role of allergy. This is particularly true in those instances where there is either symptomatic or actual pathology in the pharynx.

There is little emphasis placed on the value of local treatment.

Surgery is important. This must be thorough. Residual tonsillar tabs may be more infective and produce more symptoms than a whole tonsil. This holds true also for the adenoids. All islands of lymphoid tissue must be removed from the pharynx and the nasopharynx.

Finally, radiation is recommended, especially after incomplete surgery or where there has been a recurrence of the lymphoid or tonsillar tissue.

PERSKY, Philadelphia.

TREATMENT OF EPITHELIOMAS OF THE PALATINE TONSIL. P. C. HUET, J. LABAYLE and J. VILLEDROUIN, Ann. oto-laryng. 74:1 (Jan.-Feb.) 1957.

The authors present a series of 290 epitheliomas of the palatine tonsil from the Institute Gustave-Roussy, of which 235 were treated; 40, or 18%, were either cured or lived beyond the three-year period.

Their results depended on a number of conditions: 1. The macroscopic appearance—pedunculated, ulcerated, or infiltrated. 2. Microscopic findings—spinous cell or undifferentiated. 3. Extension of the lesion—(*a*) limited to the tonsil and only 2 cm. in size and without any apparent glandular involvement; (*b*) where the tumor is freely moveable and may or may not involve some part of the soft palate or the pillars; (*c*) where the tumor has extensive infiltration to the neighboring structures (tongue, palate etc.), and (*d*) extensive involvement in all the tissues with infiltration and ulceration. 4. The degree of adenopathy. 5. The location of the tumor.

In Group 1—small lesions with or without a minimal adenopathy—radiation therapy produces the more favorable results. This may be combined with coagulation dissection of the tonsil. In Stage 2 or 3 radiation therapy plus radium needle implantation is recommended. In this group, if the adenopathy is small and discrete, radiation alone may suffice, but if this is extensive and adherent, excision of the entire chain (after a course of radium therapy) is recommended.

PERSKY, Philadelphia.

Miscellaneous

USE OF THE ANTIHEMORRHAGIC FLAVONOIDS IN FACIAL SURGERY. WILSON A. SWANKER, Eye, Ear, Nose & Throat Month. 36:231 (April) 1957.

The author reports the use of a preparation called Hemocoavit in facial surgery to diminish edema and ecchymosis during and after surgery. This preparation contains the bioflavonoid factors hesperidin methyl chalcone, esculin, and rutin, as well as vitamin K and vitamin C. In elective cases two tablets are given three times daily for 10 days preoperatively. In emergencies it can be injected intramuscularly before, during, and after surgery. The injectable preparation does not contain rutin. The oral administration is continued after operation.

This regimen has been followed in 284 surgical procedures consisting of 36 rhinoplasties, 8 face lifts, 137 dermabrasions, and 103 skin grafts. Although evaluation of the results is difficult, there appears to be pronounced clinical relief of the usual postoperative symptoms and signs.

JENNES, Waterbury, Conn.

GLOSSOPHARYNGEAL NEURALGIA AND ELONGATED STYLOID PROCESS. N. ASHERSON, J. Laryng. & Otol. 71:453 (July) 1957.

Glossopharyngeal neuralgia is a disorder characterized by recurrent paroxysms of lancinating pain confined to the region supplied by the glossopharyngeal nerve, and unaccompanied by any sensory loss.

Five cases are presented which fall into this category, all of which were associated with elongation of the styloid process, and in which the presenting symptom was otalgia. All of

these cases were completely relieved of pain either by removal of the styloid tip, or by section of the glossopharyngeal nerve. A transpharyngeal approach was used in every instance, the incision being made through the posterior pillar. An external approach is also described.

Various other presenting symptoms that may be noted in elongated styloid processes include the following: 1. Pain in the throat persisting long after the tonsillectomy cavity has completely healed. This is attributed to stretching of fibrosis during healing. 2. Parasthesia, such as sensation of a foreign body. 3. Dull intermittent or persistent pain in the neck and pharynx, aggravated by swallowing.

Elongated styloid processes occur in 4% of the population, but are rarely symptomatic. For this reason such an entity should be considered as only one possibility in a differential list on referred otalgia. Some of the other more frequent causes are listed in the article.

LeJeune and Kearby, New Orleans.

Regulations for the Control of Streptococcal Infection from a Pediatrician's Viewpoint. B. B. Breese, New York J. Med. 57:3323 (Oct. 15) 1957.

Breese reports from the University School of Medicine and Dentistry, and the Rochester Streptococcal Research Group. . . . He emphasizes the relationship of streptococcal activities to rheumatic fever and nephritis. Rammelkamp has stressed adequate therapy of the primary sites of infection. The author states: "My associates and I see at least 500 bacteriologically proved streptococcal infections a year. During February and March, 1956, their laboratory reported 278 strongly positive cultures (3+ and 4+) and 128 weaker cultures. Many such infections go unrecognized, and certainly suppurative conditions in nose, throat, and ears, due to this organism, occur. Breese says that some 80% are in the throat. Any time of year, especially from January to June, one sees these cases. He recommends penicillin highly but it should be continued for at least 10 days after acute symptoms have subsided. Benzathine penicillin G in a single dose of 600,000 units is very effective but has unpleasant reactions. Broad-spectrum antibiotics are useful but seem to have more relapses than the other antibiotics. "The sulfonamides are of no value in eliminating the organism."

To summarize: The disease is extremely common, has many forms, is frequently misdiagnosed. Laboratory cultures must be determined. Children aged 4 to 8 are often attacked. Adequate therapy is obtainable. It renders the patient noninfectious rapidly and prevents complications. Recurrences and reinfections are commonplace. Siblings over 9 and under 3 years are rarely attacked.

Adults are rarely infected. Prevention of extension to siblings is not easily accomplished. As to control measures, reporting to authorities is inaccurate, incomplete, and should be abolished! Cultures must be taken and laboratory help must be demanded (from health authorities). Emphasis on control should be stressed, especially from January through June. Physicians should be urged to use penicillin or benzathine penicillin G intramuscularly, or "until a better method is found." Siblings need not be inactivated until they become ill, and prophylaxis for them is not recommended. Placarding and terminal sterilization should be necessary only in epidemics.

Abstractor's Comment: The streptococcal infections are as a rule air borne, and therefore are of great significance to the ENT specialist. We have not done our job locally in attempting to sterilize the nose and throat. Even if we cannot do it, we ought to try harder. Seemingly, pediatricians see almost all such infections, and since the antibiotics have paid sparing attention to the sources in nose and throat. Since the introduction of penicillin (all antibiotics) the ENT man sees only surgical complications and they are few. Cultures in every case of "colds" should be mandatory. We must recognize the enemy at once. Clearing of all symptoms will occur promptly, but this does not mean that the Streptococcus victim is free. Many become "carriers" without present symptoms, but may be seen later as "recurrences" or "reinfections." It is good to know that at long last, endocarditis, rheumatism, and liver and kidney conditions may be due to the ravages of streptococci (there are many "varieties" and "families"). . . . We need to go much farther into the biochemistry and bacteriology of diseases of the nose and throat. Many chronic diseases (poliomyelitis and multiple sclerosis?) may have their origin in the upper respiratory tract. This needs to be proved or disproved. To date we know all too little about "viruses," but we are learning fast. More and more of them will be recognized in days to come.

I. W. Voorhees, New York.

THE ETIOLOGY AND MECHANISM OF NUCLEAR STRIPPING IN THE ENDOCERVICAL AND ENDONASAL EPITHELIUM. E. H. ABOU-SHABANAH and J. C. ULLERY, Surg. Gynec. & Obst. 105:295 (Sept.) 1957.

Papanicolaou first described the phenomenon of nuclear stripping—nuclei without surrounding cytoplasm—in endocervical smears. He attributed this to extensive plasmolysis, the etiology of which was not fully understood. Eggston and Wolff described a phenomenon similar to plasmolysis in the nasal epithelium but did not include the evidence of the stripped nuclei. They felt that the goblet cells may fill and discharge their contents of mucigen many times before they are desquamated and thrown off as a part of the nasal secretion.

The authors described the phenomenon of nuclear stripping as it takes place in the endocervical and nasal epithelium. Their patients, numbering over 300, represented women between the ages of 20 and 43 years, plus children of both sexes, and 15 elderly men and women. Smears were prepared with Papanicolaou stains.

It was noted that both the endocervical and the nasal epithelium are identical in most respects that a few squamous epithelial cells may be found in the normal nasal smear. In both organs the cells are the columnar ciliated and the mucoid variety. The mucoid cells are of two types: the round, or cuboid, and the columnar.

The cellular activity which includes the elaboration of mucous secretion and ends by plasmolysis is independent of gonadal hormone activity but is influenced thereby. Thus, stripped nuclei are found in the nasal smears of male and female children, in elderly men and women, and the same process is also found in the cervical and nasal smears during the menstrual period in regularly and irregularly menstruating women.

Nuclear stripping, whether or not it is preceded by fragmentation, is a terminal process in the life cycle of the cell. Nuclear stripping depends on cellular activity which is present from birth until death. The nature of the intracellular secretion, however, hastens or delays the process. Under estrogenic activity the cells distend more rapidly and burst, discharging their nuclei before they are desquamated.

The factor that basically controls this continuous process all through life in males and pregnant and nonpregnant females seems at present to be neurogenic.

This article deserves perusal by rhinologists interested in nasal cytology.

FRIEDBERG, Chicago.

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A. M. A. Archives of Otolaryngology
American Medical Association
535 North Dearborn St.
Chicago 10, Illinois

News and Comment

ANNOUNCEMENTS

The Gould Award.—The Gould Award is presented annually by The William and Harriet Gould Foundation, Chicago, for outstanding research in laryngology, to encourage and reward fundamental investigations in this field. While the award recognizes the completion of a specific research project, favorable attention will be given to investigators with a long-term interest in this subject. The amount of the 1958 prize is $250. The recipient will be selected by an International Committee of Laryngologists from different geographical areas of the world. The members of the Committee are as follows:

Sir Victor Negus, 149 Harley St., London, W. 1, England

Prof. Cotohi Satta, M.D., No. 111 Morkawacho, Bunkyo—ku, Tokyo, Japan

Prof. Dr. Eelco Huizinga, Academisch Ziekenhuis, Afd. Kel-Neus-Oohelkunde, Groningen, The Netherlands

Hans von Leden, M.D., Medical Director, William and Harriet Gould Foundation, 30 N. Michigan Ave., Chicago 2, Ill.

For further information please address the nearest member of the international committee.

Home-Study Courses.—The 1958-1959 Home-Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Detailed information and application forms may be secured from Dr. William L. Benedict, the Executive Secretary-Treasurer of the Academy, 15 Second St., S. W., Rochester, Minn. Registrations should be completed before Aug. 15.

French Society of Otorhinolaryngology.—The annual meeting of the French Society of Otorhinolaryngology (Société Française d'Oto-Rhino Laryngologie) will be held in Paris from Monday, Oct. 20, to Thursday, Oct. 23, 1958. Sessions will be held in the Amphithéâtre de la Faculté de Médicine, rue de l'Ecole de Médecine, under the presidency of Professor Mounier-Kuhn.

The subjects on the program are as follows:

1. Treatment of Cancer at the Base of the Tongue. This subject will be discussed by Drs. Huet, Gignoux, Berard, Andre, and Labayle.
2. Cortisone and corticotropin (ACTH) in Otorhinolaryngology. This subject will be discussed by Drs. Aubin, Terracol, and Guerrier.

Further information may be obtained by addressing the General Secretary, Dr. H. Guillon, 6, Avenue Mac-Mahon, Paris.

American Physicians Fellowship Tour to Israel.—The American Physicians Fellowship, Inc., for the Israel Medical Association is sponsoring an all-inclusive tour to Israel for the Fourth World Medical Assembly of the Israel Medical Association, to be held in Tel Aviv, Haifa, and Jerusalem, Aug. 12-14, 1958. Tour group will depart from New York via El Al Israel Airlines on Aug. 9 and will leave Israel on Aug. 24.

For further details contact the National Office of the American Physicians Fellowship, Inc., 1330 Beacon St., Brookline 46, Mass.

Books

BOOK REVIEWS

La broncologia delle suppurazioni pulmonari. By Mario Romagnoli. Price, not given. Monograph from the Department of Otolaryngology, University of Milano. Publisher: Instituto per la diffusione di opere scientifiche, Milan, Italy, 1951.

This work is one of the long list of monographs originating from the department of Professor Pietrantoni, who, in the preface, states that the therapy in question was systematized around 1916 in the United States.

A historical survey is followed by exhaustive instructions regarding diagnosis and therapeutic technique as applied in the department at the University of Milan. The differential diagnosis between lung abscess and pulmonary suppuration of a focal nature is carefully evaluated.

Abscess formation in neoplastic conditions and around foreign bodies is discussed; this is followed by ample documentation of endobronchial antibiotic application.

Further main divisions take up the question of bronchiectasis, chronic bronchitis, complications and suppurative sequelae of pulmonary tuberculosis, suppurations in children. Case histories illustrate the text everywhere; 75 such "documents" are given and are listed, together with the respective diagnosis, in a separate index.

It is encouraging, for the laryngologist, that all branches of diagnosis and manipulations around these conditions may remain safely in his hands.

No similar monograph is known within the laryngological literature, so it was desirable to call attention to this important contribution in spite of the publication date—1951. The extraordinary amount of material is sifted with a sure hand and is presented and edited in exemplary form and quality. It should give welcome support to everybody who has to deal with related problems.

The Human Ear Canal. By Eldon T. Perry, M.D. Price, $4.75. Pp. 116, with 40 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1957.

Specialization has reached a high degree to be sure, but one may be amazed to find a scientific study of the external ear only. This is so likely to have short shrift in "the literature" devoted to general diseases. It is said that of all aural conditions which are seen by the general practitioner, some 80% are external. To him they may seem trivial, but to the patient they are vexatious, especially where pain is severe. To the patient any pain in or around the ear always brings up the word "mastoiditis," which is greatly feared. In these latter days one sees few infections in the mastoid which require surgery. The antibiotics have taken care of a surprising number, but without surgery the hearing may be seriously affected. Differential diagnosis is of prime importance.

This monograph of some 116 pages deals with the scientific approach, that is, with the anatomy and physiology of the external auditory meatus, its innervation and microbiology. Even the chemistry of cerumen is discussed! This is all to the good, but one could wish for a little more therapeutic regimen. A patient with furunculosis is suffering severe pain and wishes immediate relief. What to do? We are told here only in rather vague terms. In fact there are only four minor references to the condition. Should one incise? What about wet dressings? Or "packs"? Or systemic relief of pain? Morphine? These are the items the physician wishes to have at hand or in mind.

A good point is raised as to the coexistence of some general disease such as diabetes. Therefore the author is quite correct in demanding a thorough history and a general physical check-up. There is often more to these external aural manifestations than meets the eye. After giving immediate care and relief, the physician has just begun his task. Recurrences will intensify this viewpoint.

Fenestration of the Labyrinth: A Bibliography. By George Kelemen, M.D., Boston. Price, not given. Pp. 18. Psycho-Acoustic Laboratory, Memorial Hall, Harvard University, Cambridge, Mass., Feb. 1, 1954; Revision, Sept. 15, 1957.

This bibliography incorporates and extends two previously published bibliographical articles that appeared in other journals. It covers the period up to and including 1946. It is a most comprehensive bibliography, which should prove of inestimable value to persons reviewing this literature.

The Development and Disorders of Speech in Childhood. Muriel E. Morley, B.Sc., F.C.S.T. Price, $9.00. Pp. 440, with many charts, tables, and other illustrations. E. & S. Livingstone, Ltd., 16 and 17 Teivot Place, Edinburgh 1; Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1957.

This is an interesting book by Miss Muriel Morley, who is one of the leading British authorities in the field of speech pathology. The first section of the book presents data from a study of the speech development of the children in 1000 families in Newcastle-upon-Tyne. These data are roughly similar to those from other surveys of speech defects made in the United States. It is significant that different investigators using different methods have arrived at quite similar results.

The remaining sections of the book consider three main types of speech defects—delayed development of speech, defective articulation (caused by various factors), and stammering (generally referred to as stuttering in the United States). There is a great deal of information here, but because of the space problem the subject of treatment is somewhat compressed.

Those concerned with the controversy over the validity of the application of the term "aphasia" to cases of retarted speech development will be interested to note that Miss Morley seems unaware that any such controversy exists. She devotes an interesting chapter to developmental aphasia, and introduces a few terms unfamiliar to the American literature.

In sum, this is a stimulating and worth-while book for those concerned with problems of speech pathology.

BOOKS RECEIVED

Ciba Foundation Colloquia on Endocrinology, Volume XI: Hormones in Blood. Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Elaine C. P. Millar, A.H.-W.C., A.R.I.C. Price, $9.00. Pp. 416, with 74 illustrations. Little, Brown & Company, 34 Beacon St., Boston 6. Published in England by J. & A. Churchill, Ltd., 104 Gloucester Place, London, W. 1., 1957.

* **Medical Department, United States Army: Surgery in World War II: Ophthalmology and Otolaryngology.** Editor in Chief, Col. John Boyd Coates, Jr., MC; Editor for Ophthalmology, M. Elliott Randolph, M.D.; Editor for Otolaryngology, Norton Canfield, M.D.; Associate Editor, Elizabeth M. McFetridge, M.A. Price, $5.00. Pp. 605, with 149 illustrations (including charts and tables). Prepared under the direction of Major General S. B. Hays, The Surgeon General, United States Army, by The Historical Unit, United States Army Medical Service. For sale by the Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C., 1957.

* **The Human Ear Canal.** By Eldon T. Perry, M.D. Price, $4.75. Pp. 116, with 40 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1957.

* **A. M. A. Scientific Exhibits, 1957.** Edited by Editorial Committee, Council on Scientific Assembly, American Medical Association (various contributors). Price, $15.00. Pp. 480, mostly illustrations. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1957.

* **Tumor Surgery of the Head and Neck.** By Robert S. Pollack, M.D., F.A.C.S. Price, $5.00. Pp. 101, with 112 illustrations. Lea & Febiger, 600 S. Washington Sq., Philadelphia 6, 1957.

* **Practical Allergy.** By M. Coleman Harris, M.D., F.A.C.P., and Norman Shure, M.D., M.S., F.A.C.P. Price, $7.50. Pp. 471, with 25 illustrations and 18 tables. F. A. Davis Company, 1914-16 Cherry St., Philadelphia 3, 1957.

* **Surgery of Head and Neck Tumors.** By Hayes Martin, M.D. Price, $18.50. Pp. 430, with 600 illustrations. Paul B. Hoeber, Inc. (medical book department of Harper & Brothers) 49 E. 33d St., New York 16, 1957.

* To be reviewed.

* **Audiology.** By Hayes A. Newby. Price, $6.00. Pp. 342, with 50 illustrations. Appleton-Century-Crofts, Inc., 35 W. 32d St., New York 1, 1958.

* **Handbook of Noise Control.** Edited by Cyril M. Harris (46 contributors). Price, $16.50. Pp. 1042, with 808 illustrations. McGraw Hill Book Company, Inc., 330 W. 42d St., New York 36, 1957.

* **Fortschritte der Kiefer- und Gesichts-Chirurgie.** Volume 3. A Yearbook. Edited by Prof. Dr. Dr. Karl Schuchardt. Price, $22.85. Pp. 359, with 391 illustrations. Georg Thieme Verlag, Herdweg 63 (14a) Stuttgart (American agent—Grune & Stratton, Inc., 381 Fourth Ave., New York 16). 1957.

Nomina Anatomica. Vergleichende Uebersicht der Basler, Jenaer und Pariser Nomenklatur. Fifth Edition. By Prof. Dr. Fr. Kopsch. Revised by Prof. Dr. Dr. K.-H. Knese. Price, $1.55. Pp. 155. Georg Thieme Verlag, Herdweg 63 (14a) Stuttgart (American agent—Grune & Stratton, Inc., 381 Fourth Ave., New York 16), 1957.

* **Headache: Diagnosis and Treatment.** Second Edition. By Robert E. Ryan, B.S., M.D., M.S. (in Otolaryngology), F.A.C.S. Price, $6.75. Pp. 421, with 6 illustrations. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1957.

Sprachaudiometrie: Grundlagen und Praktische Anwendung Einer Sprachaudiometrie für das Deutsche Sprachgebiet. By Doz. Dr. Med. Karl-Heinz Hahlbrock. Price, $5.00. Pp. 182, with 123 illustrations. Georg Thieme Verlag, Herdfek 63 (14a) Stuttgart (American agent—Grune & Stratton, Inc., 381 Fourth Ave., New York 16), 1957.

Radium-Isodosen: Die Radiumdosierung in "r" (German and English texts). By Dr. Med. August Verhagen. Price, $4.65. Pp. 103, with 37 illustrations. Georg Thieme Verlag, Herdfeg 63 (14a) Stuttgart (American agent—Grune & Stratton, Inc., 381 Fourth Ave., New York 16), 1958.

Les Bourdonnements d'oreille. By A. Appaix, J. Bouche, and G. Brémond. Price, 2000 francs. Pp. 240, with 31 illustrations. Librairie Arnette, 2, rue Casimir Delavigne, Paris, 1957.

Les Tumeurs malignes de l'oreille (oreille interne exclué). By J. Leroux-Robert and A. Ennuyer. Price, 3000 francs. Pp. 336, with 122 illustrations. Librairie Arnette, 2, rue Casimir Delavigne, Paris, 1957.

Année Oto-rhino-laryngologique, 1957. By A. Aubin and M. Bouchet. Price, 1500 francs. Pp. 224, with 112 illustrations. Librairie Arnette, 2, rue Casimir Delavigne, Paris, 1957.

* **Noise and Your Ear.** By Aram Glorig Jr., M.D. Price, $6.50. Pp. 152, with 31 illustrations and 6 tables. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16; 99 Great Russell St., London, W. C. 1, 1958.

* To be reviewed.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.
Place: Nova Scotian Hotel, Halifax, N. S. Time: June 9-11, 1958.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba.
Meeting: Third International Congress of Allergology.
Place: Paris. Time: Oct. 19-26, 1958.

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh International Congress of Bronchoesophagology.
Place: Kyoto, Japan. Time: Sept. 12-14, 1958.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. E. G. Gill, 711 S. Jefferson St., Roanoke, Va.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: Rio de Janeiro, Brazil. Time: Aug. 10-16, 1958.
President of Congress: Prof. R. David de Sanson.
Secretary of Congress: Dr. Walter Benevides, Caixa Postal 2838, Rio de Janeiro.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Alden Miller, 500 S. Lucas Ave., Los Angeles.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco. Time: June 23-27, 1958.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Annual Meeting: Palmer House, Chicago, Oct. 12-17, 1958.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Walter Hoover, 605 Commonwealth Building, Boston.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

American Laryngological Association

President: Dr. Harry P. Schenk, 326 S. 19th St., Philadelphia 3.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

SECTIONS:

Eastern.—Chairman: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Southern.—Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. Francis L. Lederer, 860 N. Lake Shore Drive, Chicago.
Western.—Chairman: Dr. Ben. R. Dysart, 960 E. Green St., Pasadena 1, Calif.

American Rhinologic Society

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, October, 1958.
Annual Meeting: Chicago, October, 1958.

American Otological Society, Inc.

President: Dr. Dean M. Lierle, State University of Iowa, Iowa City.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: The Homestead, Hot Springs, Va. Time: 1959.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

American Society of Facial Plastic Surgery

President: Dr. Trent W. Smith, 327 E. State St., Columbus 15, Ohio.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.
Meeting: July 23, 1958, place to be announced.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.
Place: Palmer House, Chicago. Time: Oct. 16-17, 1958.

Otosclerosis Study Group

President: Dr. Joseph A. Sullivan, 174 St. George St., Toronto 5, Canada.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Building, Louisville.

Society of Military Otolaryngologists

President: Capt. William C. Livingood, U. S. N. (MC).
Secretary-Treasurer: Lt. Col. Stanley H. Bear, USAF(MC), 3810th USAF Hospital, Maxwell AFB, Alabama.

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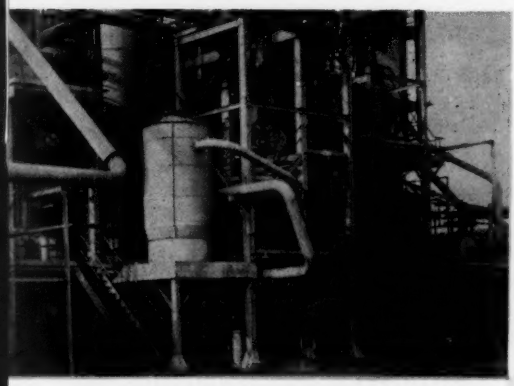

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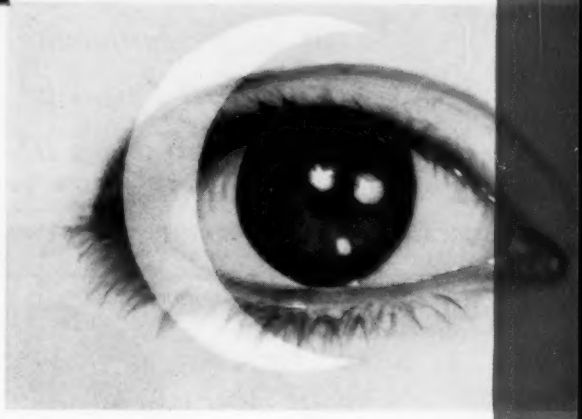

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2. Kuhn, H. S.: Tr. Am. Acad. Ophth. *55*:431, 1951.

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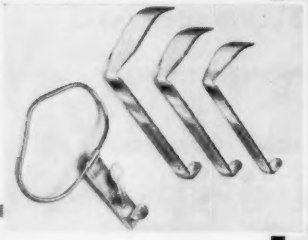

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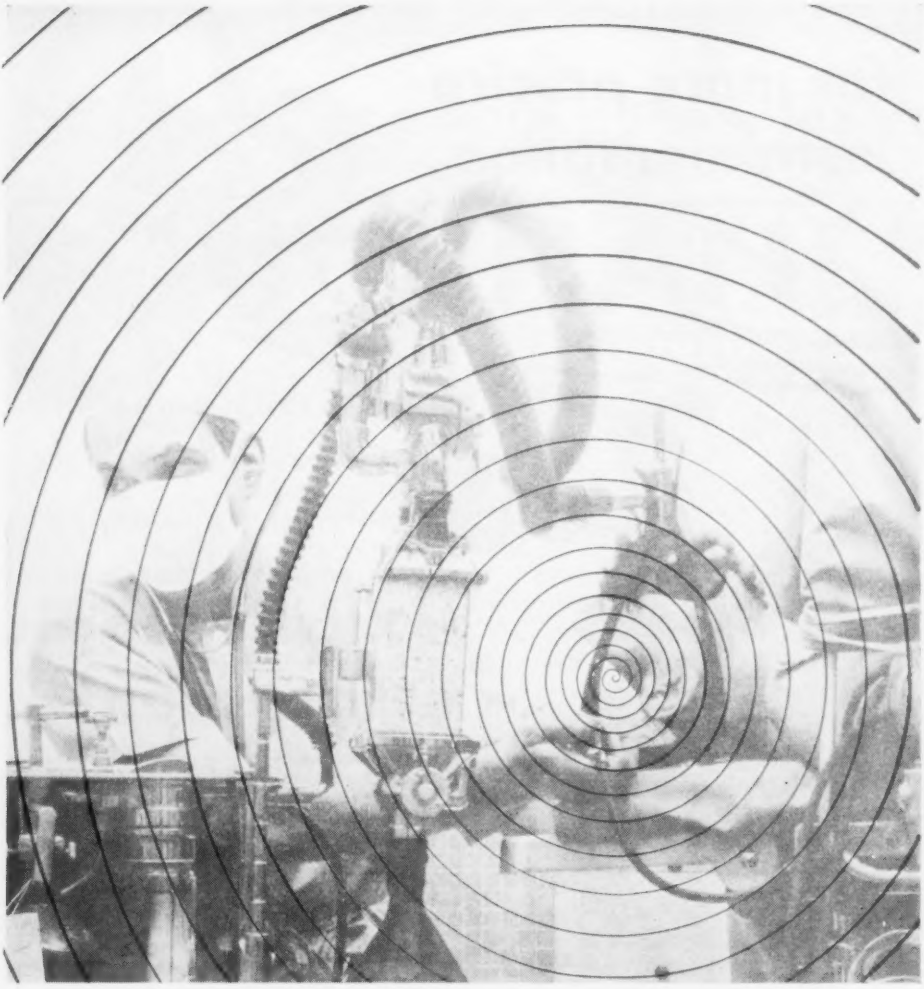

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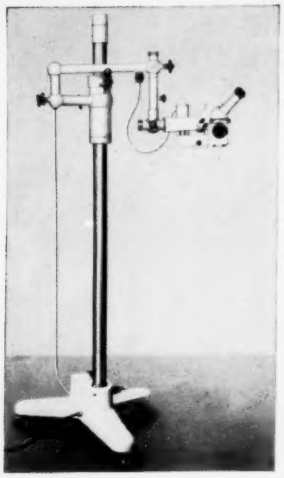

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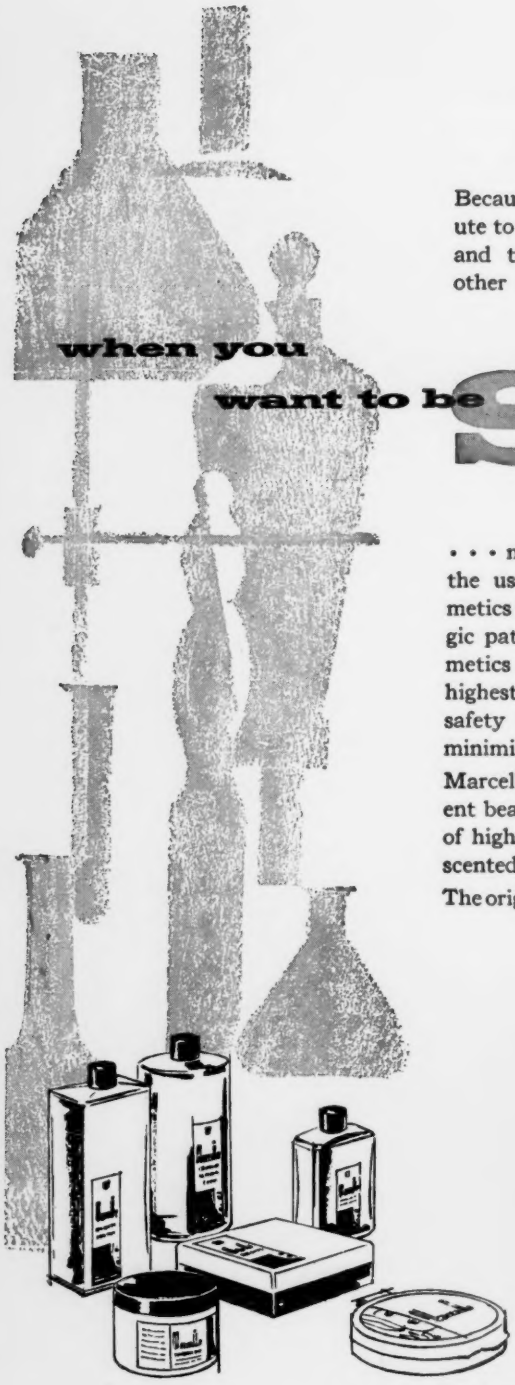

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